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Medicine

Hirsutism in the Female*

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Hirsutism in the female has been defined as abnormal growth and distribution of hair. Many women complain of this distressing condition, particularly when the face is involved. Although medical treatment is frequently unsuccessful in removing the excess hair some of the causative lesions are curable. As there is a morbidity and mortality associated with these lesions, it is important that the cause of the hirsutism be found. We intend to discuss the differential diagnosis of hirsutism and present several case reports as examples. A suggested plan for investigation of the hirsute female is also presented.

Human Hair Growth

Our knowledge of the physiology of human hair is incomplete. Danforth[5] has reviewed the subject. He divides human hair into three types: 1) lanugo or general body hair, which is probably uninfluenced by endocrine factors; 2) the ambosexual hair of males and females, which is dependent on hormone stimulation and which, while present in both sexes, differs in amount and pattern (pubic and axillary hair are examples of this type); and 3) true sexual hair, as represented by the beard of the male.

Callaway et al[4] feel that there are three main factors influencing hair growth: (a) constitutional, the ability of any individual follicle to respond to endocrine stimulation; (b) hormonal, the stimulus for hair production to those follicles specifically sensitive to hormonal action; and (c) local, the nutrition and the blood and nerve supply of the follicle. Malnutrition is usually assocated with decreased rate of hair growth. However, while obesity and hirsutism often co-exist, there does not seem to be any causal relationship.

Classification of Hirsutism

Several classifications of hirsutism have been proposed. Table I shows the anatomico-pathologic classification of Greenblatt[7] modified to include more ovarian lesions. A separate heading to indicate placental causes has been suggested by Benson et al[1]. Table 2 shows a clinical classification proposed by Benson[1]. In this classification an attempt is made to differentiate sharply between defeminization and virilization. This differentiation is largely a quantitative one. de Alvarez[6] also uses a clinical classification: hirsutism with a virilizing endocrinopathy, with a non-virilizing endocrinopathy and with no recognizable endocrine disorder.

Hirsutism with a Virilizing Endocrinopathy

1. **The adrenogenital syndrome** is the result of overproduction of androgenic adrenocortical hormones. If the stimulus is effective in the early fetal period pseudoprecocious puberty occurs in the male and hermaphrodism in the female[9]. It is beyond the scope of this paper to deal with these interesting and frequently fatal conditions. With post-pubertal onset in a female, hirsutism, amenorrhea, enlargement of the clitoris[2], lowered pitch of the voice and increased muscle development are the most frequent signs and symptoms. Because of the over-production of adrenal androgen the 17-ketosteroid excretion in the urine is increased. Differentiation of the syndrome into its three common causes of hyperplasia, adenoma or carcinoma can be made by a series of suppression tests, steroid pattern differences[3], intravenous pyelography, retroperitoneal pneumography, and perhaps exploratory laparotomy. Hyperplasia is best treated by cortisone in replacement dosages. An adenoma or carcinoma should be removed surgically.

2. **Arrhenoblastoma of the Ovary** is usually a benign unilateral neoplasm. The presenting symptoms and signs are identical with the adrenogenital syndrome. An ovarian mass is usually palpable on pelvic examination. The urinary excretion of 17-ketosteroids is usually normal. Surgical excision cures the condition. The other ovarian masculinizing lesions are even more rare, present in the same way and respond well to surgical removal.

Hirsutism with Nonvirilizing Endocrinopathy

1. **The Stein-Leventhal Syndrome,** amenorrhea associated with bilateral polycystic ovaries, was first described in 1935[18]. In order of frequency, presenting symptoms are (a) amenorrhea; (b) sterility; (c) hirsutism; (d) pain; (e) excessive uterine bleeding[6].

The ovaries are usually 4 to 6 cm. in diameter, oval, grey in color, with a thickened capsule. They are firm and relatively insensitive to pain; they feel smooth rather than nodular.

All degrees of hirsutism may be found. In half the patients, breasts are smaller than normal. Characteristically, puberty and early adolescence will be normal or nearly so; after a few years, periods of amenorrhea increasing in length, occur and hair growth becomes excessive on the face, abdomen and limbs.

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The most valuable diagnostic sign is the finding of bilateral smooth enlarged ovaries. Urinary 17-ketosteroids and 17-hydroxysteroids are usually normal; the former may be slightly raised.

The accepted treatment of this syndrome is bilateral wedge resection of the ovaries. Leventhal[10] discusses 114 patients who had this operation; 86% became pregnant during the follow-up period. Perloff et al[14] have postulated adrenocortical hyperfunction as the etiological mechanism in this syndrome and claim significant regression of hirsutism following treatment with cortisone. Other workers have combined surgery and hormone therapy.

2. **Cushing's Syndrome** is considered to be caused by excessive production by the adrenal cortex of hydrocortisone. Here, in contrast to the adrenogenital syndrome, the defect is concerned with the hormones associated with gluconeogenesis, not the androgenic steroids. Some patients, however, show components of both diseases.

The characteristic clinical features of the syndrome are obesity, hypertension, gonadal dysfunction, decreased glucose tolerance, fatiguability and muscular weakness. Hirsutism of the face and chest and male pubic escutcheon are commonly seen. Baldness is uncommon. The obesity is confined to the face and trunk and there is often a "buffalo-hump" of fat. Purplish striae on the abdomen, thighs and breasts are also found. The clitoris is not enlarged in "pure" forms of the syndrome.

"The signs and symptoms that aid in the differentiation of 'pure' Cushing's syndrome, 'pure' adrenogenitalism and the Stein-Leventhal syndrome are that in the first there is muscular weakness and pseudomasculinization, in the second there is increased strength and true virilism and in the third there is hirsutism with marked femininity"[6].

The urinary excretion of 17-hydroxysteroids is usually significantly increased in this syndrome. Excretion of 17-ketosteroids may be normal or moderately increased.

Surgical therapy consists in removal of a tumor if present. If hyperplasia is found, total adrenalectomy is now considered to be the therapy of choice, with life-long cortisone replacement.

3. **Achard-Thiers Syndrome,** "the bearded lady with diabetes," is considered by some to be a distinct entity[17] but most workers now believe that the association is fortuitous[6].

Hirsutism with no Recognizable Endocrine Disorder

1. **Idiopathic Hirsutism** is the most common type of hirsutism in women. de Alvarez suggests that an altered end-organ response to normal circulating androgen levels may occur in these patients[6].

Historically, hirsutism has usually been present from puberty and seldom shows sudden spurts of growth. A familial history is often obtainable. Excepting the hirsutism itself, physical signs are usually lacking, so that there is no evidence of masculinization.

Most workers report normal or slightly elevated 17-ketosteroid excretion. Perloff et al[13, 15], however, have fractionated the 17-ketosteroids into a $C_{19}O_2$ group (poorly androgenic) and a $C_{19}O_3$ group (very strongly androgenic) and found the latter fraction consistently above normal. Although some of the patients reported in this series are probably examples of the Stein-Leventhal syndrome, the findings are suggestive that a definite endocrine dysfunction can be measured in patients with idiopathic hirsutism. Perloff and his coworkers[13, 15], claim considerable success in treating this syndrome with prednisone.

2. **Hirsutism Medicamentosa** has been reported to be related to therapy with a variety of therapeutic agents. Testosterone is probably the most frequent agent involved[6, 12]. Hirsutism with Dilantin therapy has also been described[2, 11]. Often many months elapse after cessation of therapy before the excess hair disappears.

3. **Miscellaneous:** Hirsutism is associated with a variety of conditions, including encephalitis (see case report below), traumatic shock[16], burns[2] and as the only clinical manifestation of adrenal adenoma[8].

Case Reports

1. **Mixed Adrenogenital Cushing Syndrome.** A 28 year old single female was seen because of hirsutism, weight gain and amenorrhea all starting about the age of 23 (Fig. 1). Her father is very hirsute

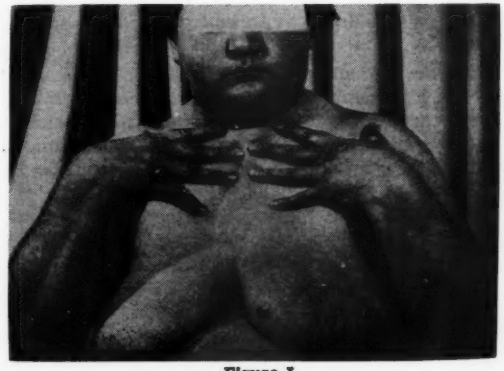

Figure I
Case 1: Heavy growth of hair on the arms is well shown here, as well as hair on the breasts

as are three brothers. She has one sister who has a normal hair distribution. There was no evidence of muscle wasting, nor breast atrophy. The clitoris was enlarged. Obesity was generalized. The facial hair growth was heavy and black, requiring shaving every other day. The anterior chest wall, breasts and abdominal wall showed a male distribution of hair. The blood pressure was 210/130. There was an ulcer produced by minimal trauma on the left leg which had not healed. There was no

hematological nor biochemical evidence of Cushing's syndrome. The 17-ketosteroid excretion was slightly raised and the 17-hydroxysteroid excretion was normal.

Radiological examination of the sella turcica showed slight enlargement. An intravenous pyelogram was normal and retroperitoneal pneumography did not reveal a definite tumor.

Because of the severe hypertension and the non-healing ulcer, it was felt that this was a mixed adrenogenital-Cushing syndrome. A subtotal adrenalectomy was performed after examining the ovaries. Menses restarted, but the hypertension persisted. Re-investigation after cortisone withdrawal revealed that her androgen excretion was still high. A second laparotomy was performed and a normal appearing left adrenal was found and removed. Again the hypertension persisted and the 17-ketosteroid excretion was still elevated. ACTH stimulation showed a very rapid increase in steroid excretion so a third laparotomy was performed and an adrenal was found on the right side and removed. She is now maintained on 37.5 mgm cortisone with a decrease in the hypertension and decrease in hair growth.

This case illustrates the overlapping picture frequently found between Cushing's syndrome and adreno-genitalism. Adrenalectomy is not recommended for adreno-genitalism. This patient was operated on because of the severe and progressive hypertension.

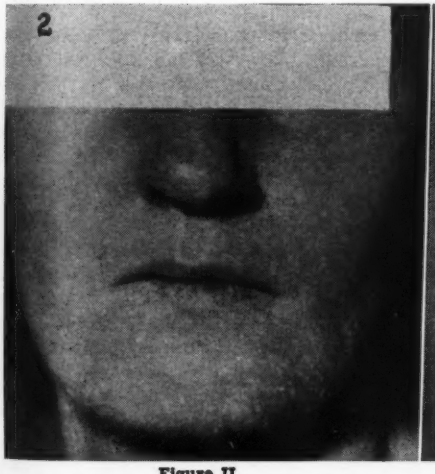

Figure II
Case 2: Note the profuse growth of facial hair; also hair on the neck

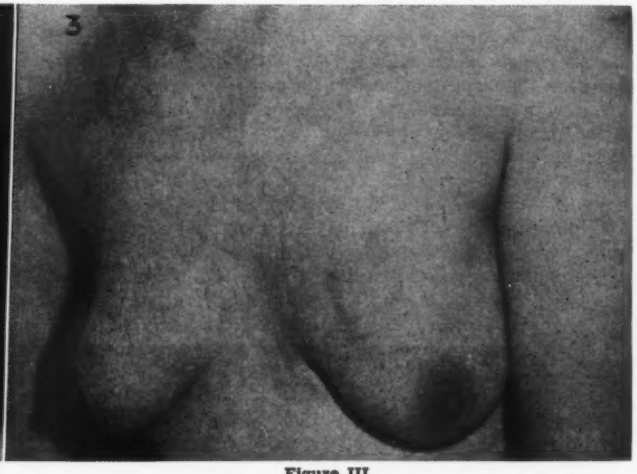

Figure III
Case 2: Circum-areolar and pectoral hair, with essentially normal breasts

2. **Stein-Leventhal Syndrome.** This 29 year old married woman was admitted to hospital in January, 1959, complaining of a recent marked increase in growth of facial and body hair and amenorrhea since August, 1958.

Her menarche was at 15; the intermenstrual period varied from 21 to 28 days, with a moderate flow lasting three days. In May, 1958, she delivered a term infant following a normal pregnancy and labor. Following delivery her milk came in, but was insufficient in quantity. Her menses resumed in June and she had normal periods in July and August.

The patient had always had some hair on her face, neck, breasts, chest and on the arms and legs. She noticed a sudden increase in hair growth after her baby was born in May, with an increase in the rate of growth, number of hairs, coarseness of hairs, with deepening colour. The pubic hair, which previously grew part way to the umbilicus, was now growing above it.

On examination, the patient was of definitely feminine habitus, with a heavy growth of long hair on the cheeks, chin, upper lip, arms and legs, between and on the breasts and on the abdomen (Fig. 2 and 3). The breasts were within normal limits for size.

No abnormal findings were noted, aside from excessive hair growth, on examination of the head and neck, chest, abdomen and extremities. The blood pressure was 110/80. On vaginal examination the right ovary was just palpable. There was a normal anteverted uterus; the clitoris was not enlarged.

No abnormalities were found on routine examination of blood and urine. A Friedman test was negative. The glucose tolerance test was normal. Urinary 17-ketosteroids were 7 mgm/24 hours (normal 5-15 mgm/24 hours); 17-hydroxysteroids were 4 mgm/24 hours (normal 6-19 mgm/24 hours). Total esinophils were 211 per cubic millimetre of blood.

A laparotomy was carried out. The right ovary was 7 x 2 cm., the left 3 x 2 cm.; both ovaries had a firm white capsule with no evidence of ovulation. A soft cystic swelling was present at the outer end of the right ovary. Wedge resection was performed

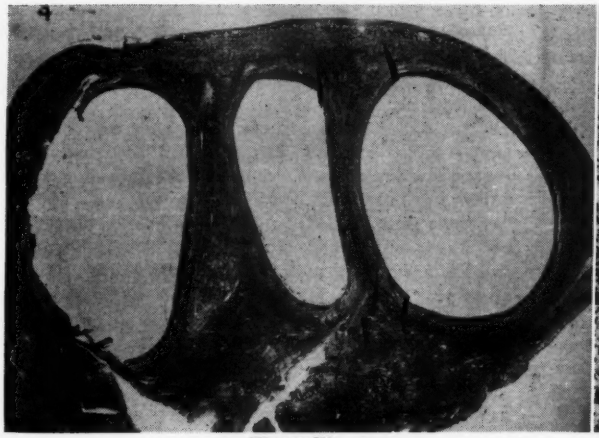

Figure IV
Cross section of ovary showing dilated Graafian follicles (H & E X12)

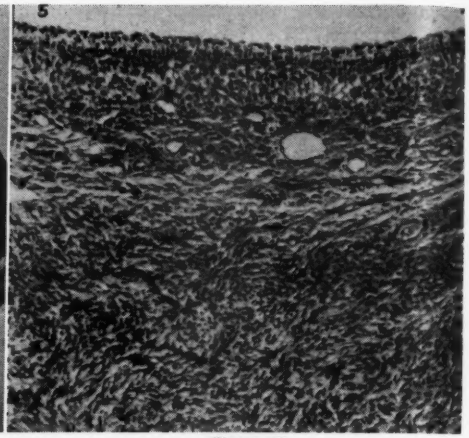

Figure V
Wall of Graafian follicle. The inner lining is composed of granulosa cells; the thecal layer is hyperplastic (H & E X110)

on both ovaries. The biopsy specimens were found to contain multiple follicular cysts in both ovaries, with thecal hyperplasia; the findings were compatible with the diagnosis of Stein-Leventhal syndrome (Fig. 4 and 5).

Two days after her operation, the patient's menses resumed.

This case demonstrates the classic clinical findings of hirsutism (without masculinization), amenorrhea, and a palpable ovarian mass, which is typical of the Stein-Leventhal syndrome[10, 18]. The presence of this syndrome in a woman who has previously had a child is not contradictory and has been described before. One of Stein's original seven patients had had a baby prior to the onset of the syndrome[18].

Although the majority of patients operated upon resume normal menses and produce children, most have little improvement in their hirsutism.

3. **Idiopathic Hirsutism.** This 31 year old married woman was admitted to hospital in February, 1959. Her complaints were of excessive weight gain and increased hair growth. When married, 13 years ago, she weighed 105 pounds; after three months she became pregnant, and during the next nine months gained 87 pounds. After delivery her weight remained in the neighborhood of 190 pounds, except when dieting.

In September, 1958, a sister noticed that the patient had more than the usual amount of hair on her face, with a small mustache. The patient noticed a change in her pubic hair pattern; a thin line of hair was growing up towards the umbilicus. There was also more hair on the lower limbs and in the axilla.

The patient's mother and father are normal in weight. She has six brothers, whose weights vary from 120 to 220 pounds; and seven sisters, one of whom weighs 300 pounds. None of the immediate relatives is particularly hirsute.

Her menarche was at age 8. Her periods occur at 28 day intervals, with a moderate flow lasting 5-6 days; she has mild dysmenorrhea. She has two children and has had three miscarriages.

On physical examination, the patient was very obese, with a full, flushed hirsute face. The breasts were pendulous. Blood pressure was 125/90. The abdomen was obese, protuberant and showed hair growing upward from the supra-pubic area as in the male. The skin showed some acne. The limbs were thick and had strong muscles.

Routine laboratory examination of blood and urine was normal. X-rays of the chest and skull were normal. The urinary excretion of 17-ketosteroids and 17-hydroxysteroids were normal, as was a glucose tolerance test and I^{131} uptake. A vaginal smear showed evidence of estrogen activity.

The final diagnosis in this case was idiopathic hirsutism. No evidence of endocrinopathy was found. In the previous case, of Stein-Leventhal syndrome, the laboratory investigation was also non-contributory, but the findings of amenorrhea and a palpable ovarian mass were considered to be adequate indications for laparotomy. No such indications were present in this case.

4. **Hirsutism Associated with Encephalitis.** This 9 year old child was born in December, 1949. Her birth weight was five pounds, eleven ounces. She required some resuscitation but thereafter did very well. She sat up at six months of age and could walk with support at nine months. Then, at 9 months of age, she developed encephalitis. She was ill for a month. Since the attack of encephalitis she has been totally disabled. She is unable to see or speak, but can hear. She cannot walk or perform co-ordinated movements. She has been admitted to hospital with frequent bouts of pneumonia in the ensuing years.

She has generalized spasticity. Clonus is present in the upper and lower limbs; she also has flexor

spasm of the upper limbs and extensor spasm of the lower limbs. There is generalized hyperreflexia. The Babinski response is present bilaterally. She has both grand mal and Jacksonian type convulsions.

When the child was three years old her menarche occurred. She received x-ray therapy to her abdomen and menses ceased for nearly six years, recommencing in the summer of 1958.

On one admission, in 1953, age 4, the following comment was recorded: "She looks to be 15 years old. Has pubic hair; started to menstruate when she was three years old. Has thick hairy thighs and legs and a little bit of breast tissue." Later the same year "marked hypertrophy" of the breasts was noted.

This case does not represent true hirsutism, but is a case of precocious puberty following severe encephalitis. The growth of hair, while abnormal for the age of the patient, was in the normal distribution for a pubertal girl.

Diagnostic Measures

These can be summed up as follows[6]: A good history and physical and pelvic examination are the sine qua non of diagnosis of hirsutism in women. Particular attention should be paid to: age of onset, family history of hirsutism, history of menstrual irregularities, voice changes, development of acne and baldness, change in breast size, change in character and distribution of hair, libido, sterility, pregnancy, presence or absence of other disease, drug medication, etc. The physical examiner should note: general condition of patient, distribution and texture of hair, body build, fat distribution, muscular strength and development, appearance of skin, presence or absence of baldness, pelvic or abdominal masses, size of clitoris, size, position and texture of other sex organs, size of larynx and sound of voice.

The findings of the history and physical examination, outlined above, should determine what laboratory, x-ray and other investigation should be carried out in any individual case. Estimation of the urinary excretion of 17-ketosteroids should be performed in every patient having hair growth differing significantly from the familial pattern, particularly where this growth of hair does not coincide with puberty or the menopause. In most patients in this group the results will be in the normal range and no further investigations need be performed. If the excretion of 17-ketosteroids is increased, suspicion must be directed at the adrenal gland.

When hirsutism is accompanied by other signs and symptoms, the appropriate organ must be investigated. X-rays of the skull, intravenous pyelogram and pneumography with pre-sacral air insufflation all have their role. Examination of the pelvis under general anaesthesia may be necessary, particularly in the nulliparous female. Urinary 17-hydroxysteroids should be determined along with the 17-ketosteroids.

Finally, laporotomy may be considered. Weed[10] recommends laparotomy with exploration of the adrenal areas and bilateral ovarian biopsies whenever there is strong suspicion of an endocrinopathy, if a satisfactory diagnosis cannot be made. This principle was followed in the second case recorded above; a presumptive diagnosis of Stein-Leventhal syndrome was verified at operation.

Acknowledgments

We would like to thank Doctors R. O. Burrell, M. B. Wall, P. Goldstein and H. L. Davies who allowed us to study these patients. We would also like to acknowledge the assistance of Miss Charlotte Stankiewicz, Medical Records Office, Mr. L. Stanford, Art and Photography Department and Doctor M. Van Wijhe, Department of Pathology, in the preparation of this paper.

Table I

Anatomico-Pathologic Classification of Hirsutism. Adapted from Greenblatt[7]; additions are bold face type.

1. Pituitary:
   a) Cushing's Disease
   b) Achard Thier's Syndrome (?)
   c) Acromegaly
   d) Syndrome of excess L.H. (??)
2. Adrenal:
   a) Tumor (i) adrenogenital syndrome
      (ii) Cushing's syndrome
   b) Hyperplasia: (i) congenital — pseudohermaphrodism
      (ii) post-natal — adrenogenitalism
      (iii) Cushing's Syndrome
3. Ovary:
   a) Tumor: (i) arrhenoblastoma
      (ii) adrenal rest tumor
      (iii) **hilar cell rest**
      (iv) **gynandroblastoma**
      (v) **dysgerminoma**
   b) Ovotestis (hermaphrodism)
   c) Stein-Leventhal syndrome
4. Genetic:
   a) Racial or familial
   b) Sensitivity to intrinsic androgens
5. **Placenta:**
   a) **Hirsutism of pregnancy**
   b) **Chorionepithelioma**

Table II

Physical Findings in Hirsutism, Defeminization and Virilization Adapted from Benson et al[1]

1. "Simple" Hirsutism:
   a) Hirsutism, all degrees
   b) Amenorrhea
2. Hirsutism with Defeminization:
   a) Acne, roughening of skin, odorous perspiration
   b) Recession of hairline at forehead with slight alopecia
   c) Loss of feminine contours
   d) Partial involution of genitalia
3. Virilization, including hirsutism and defeminization:
   a) Enlargement of clitoris
   b) Coarseness of voice; enlargement of "Adam's Apple"
   c) Balding
   d) Increased musculature
   e) Atrophy of breasts and genitalia

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Hyperthyroidism: A Problem in Diagnosis in the Presence of Heart Disease

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Introduction

Hyperthyroidism usually presents little difficulty in diagnosis. Exophthalmos, thyroid enlargement (whether diffuse or nodular), nervousness, tremor, palpitation or warm, moist, flushed skin usually give the diagnosis away. There are times, however, when the diagnosis becomes a problem. It is the purpose of this paper to discuss these difficulties as they apply to patients in heart failure.

An elevated level of thyroid hormone produces effects upon the cardiovascular system. It is therefore not surprising that the signs and symptoms in this condition will mimic those of heart failure. Among the symptoms common to both processes are dyspnea, palpitation, weakness, chest pain, cough and edema[1]. Another reason for difficulty in the diagnosis of hyperthyroidism in the presence of congestive heart failure is that the signs and symptoms of thyroid disease may actually be masked by those of heart failure. The weakness and tiredness may be explained away by heart failure; the loss of tissue may be covered by edema; the increased appetite may be offset by anorexia associated with heart failure and the tachycardia and atrial fibrillation may be accepted as due to heart disease[2]. If, then, the eye signs are absent and there is no visible or palpable thyroid enlargement, the diagnosis may be completely missed[2, 3].

It has long been known that hyperthyroidism adversely affects the cardiovascular system. In the first written account of hyperthyroidism, Parry in 1786, described an apparent enlargement of the heart, dyspnea, anasarca, irregular rhythm and tachycardia, associated with a marked enlargement of the thyroid gland[4]. Since then many other authors have indicated that the heart was involved in thyrotoxicosis and even that thyrotoxicosis could, in association with other heart disease and even by itself, result in congestive heart failure[1, 2, 3, 5-11].

Despite the frequency of these findings and also the repeated warnings to physicians to suspect thyrotoxicosis as a factor in congestive heart failure, it is apparent that hyperthyroidism as a contributing factor in the etiology of congestive heart failure still presents a difficult problem. We have seen three such patients in the past two years. They constitute the raison d'être for this paper.

These three patients were treated in St. Boniface Hospital. Each had been seen on at least one occasion previously and heart disease diagnosed. Each was in heart failure when seen in this hospital and each was more or less refractory to the standard treatment of congestive heart failure. Hyperthyroidism previously missed was found to be present in each of the three cases. The case histories are presented below:

Case 1

Mrs. A. S. This 48 year old woman was first admitted to St. Boniface Hospital, January 23, 1957 complaining of ankle swelling and of dyspnea for one year, increasing in recent months. There was a history of palpitations nine years previously that had been "cured by drops" and which had recurred in association with the dyspnea and edema. The past illnesses included a left-sided hemiplegia in 1950, from which she had recovered. Her appetite was good and her bowel habits regular and she had no melena, Treatment prior to admission was with digoxin, diamox, peritrate and vitamins. On examination, the patient was of medium build, cyanotic and dyspneic. There were no eye signs and no palpable thyroid mass. The jugular venous pressure was elevated, and the pulse was hyperkinetic and irregular, with a pulse deficit of 50. The B.P. was 130/80. There were basal crepitations in her lungs. There was a diffuse thrusting apex beat in the sixth interspace at the anterior axillary line. A grade iii/iv soft blowing systolic murmur at the apex and a palpable apical systolic thrill were noted. There was pitting edema to the knees and severe sacral edema. She was diagnosed as congestive heart failure due to mitral regurgitation. Chest x-rays showed left ventricular hypertrophy, pulmonary congestion and free pulmonary effusion.

The electrocardiogram revealed atrial fibrillation and hypertrophy and ischemia of the right ventricle, while the phonocardiogram indicated mitral stenosis and incompetency. The urine contained 0.1 gm% protein and the hemogram showed a low hemoglobin (9.7 grams%) and hematocrit (35%) and an elevated sedimentation rate (47 mm). While in hospital she was treated with digoxin and thiomerin. She developed a thrombophlebitis and was anticoagulated for two weeks. She improved symptomatically but was still refractory to the treatment of her failure as evidenced by persistent dyspnea and edema.

The patient left hospital against advice February 5, 1957. A protein bound iodine estimation returned after discharge with a value of 8.1 ugm% and on re-admission the patient was still in failure. On February 26, 1957 her thyroid function was further investigated. The B.M.R. was 44% and the radioactive iodine uptake 57% in 24 hours. She still had a low hemoglobin and an elevated sedimentation rate, while her urine, serum electrolytes and B.U.N. were normal.

On re-examination there were still neither eye signs nor a palpable thyroid. In addition to previous cardiac findings a mid-diastolic murmur was heard at the apex.

She was again digitalized, given a complete battery of diuretics (mercurials, diamox, diuril and H4 CL), aminophylline and potassium chloride. 8.98 millicuries of I^{131} were given March 14 and followed by administration of Lugol's iodine for 9 days. 14.7 millicuries of I^{131} were given June 7. Between this second administration of I^{131} and her discharge July 15, 1957 the patient improved, lost her edema and dyspnea and increased her body tissue. Her weight, 129 pounds on admission dropped to 108 pounds with loss of her edema and rose to 124 pounds with increase in appetite and body tissue. An attempt has been made to follow this woman, but she had not reported to the Out-Patients' Clinic at the time of writing.

Case 2

Mrs. H. J. This 77 year old woman was admitted to St. Boniface Hospital April 30, 1958 with post-prandial distention and discomfort of six months duration, dyspnea on exertion, intermittent claudication and a weight loss of 14 pounds in one year. Diuretic therapy had been instituted before admission. Her past history had included removal of her gall-bladder, uterus and appendix.

On examination she was an elderly dyspneic lady who had neither eye signs of hyperthyroidism nor a palpable thyroid. There was no elevation of jugular venous pressure, her chest was clear and there was no ankle or sacral edema. Her blood pressure was 140/80 and her pulse 72 and regular. Chest x-rays showed a prominent left heart border with an elongated aorta and calcified arch. The electrocardiogram showed ischemic changes at the apex. The hemogram, serum electrolytes, serum proteins, blood sugar and 17-ketosteroid and 17-hydroxysteroid urine excretions were all normal. She had mild proteinuria and a B.U.N. of 26 mg%. She was treated for mild congestive failure and discharged apparently well, May 7, 1958.

She was re-admitted January 26, 1959 semi-conscious and with dyspnea, a painful left hip and constipation for three days. She was on digoxin 0.25 mg. daily and following administration of sparine for her pain had become semi-conscious. Other noteworthy historical points were anorexia and a weight loss of 35-40 pounds in the last few months.

Physical examination revealed an elderly semi-conscious woman with rapid, shallow respirations who was having oculogyric crises. Her chest was dull and her breath sounds bronchial. Her blood pressure was 150/80 and her pulse 84, irregularly irregular. She had no edema. She was tender in the right upper and left lower quadrants, and her liver was palpable 2 fingers below the right costal margin. She was diagnosed as either pneumonia or congestive heart failure and a possible cerebrovascular accident. Chest x-rays were unchanged. A barium series showed a duodenal diverticulum. Abdominal films were negative. The electrocardiogram showed atrial fibrillation, ventricular extrasystoles and right ventricular hypertrophy; significant laboratory findings were as follows: Sedimentation rate, 41 mm; White blood count, 15,400 (78% polymorphonuclears); Pus cells, 50-60; Red blood cells, 40-50; Protein in urine, 0.05 gm%; B.U.N., 24.

In view of her atrial fibrillation and a barely palpable thyroid isthmus, her thyroid function was investigated although the diagnosis of hyperthyroidism was not seriously entertained. The P.B.I. was 7.4 ugm% and the I^{131} up-take was 68%. She was treated for her urinary infection and her congestive heart failure and discharged February 10, 1959. February 12, 1959 she received 5 millicuries I^{131}. She was last seen March 26, 1959 at which time she was feeling better, and was more active according to her daughter, than she had been in the past two years. She still had a palpable liver and her respirations were sighing, but she was otherwise physically well. It was only six weeks since her treatment and she had not had the full effect of her I^{131} therapy as yet. She was still taking digoxin 0.25 mg. daily.

Case 3

Mrs. M. S. This woman was seen at St. Boniface Hospital as an out-patient in October, 1957 at the age of 42. She had been subject to migraine headaches for thirty years. When they became worse and throbbing in nature, she saw a doctor who found her hypertensive. She had been able to keep up with the chores on her farm until one month previously when she became dyspneic on slight exertion and had a tightness around the neck. She had also had palpitations and edema of the legs

occasionally, but persistent in the last week. Her appetite was exceptional and she had gained 21 pounds in two weeks. She admitted to heat intolerance, and used less blankets than her husband. She had frequency, nocturia and stress incontinence of uncertain duration and had had irregular menses with flushes for four years.

On examination the patient was of medium build, florid and had warm moist extremities. Her chest was clear. B.P. 220/85, pulse 90 with occasional extrasystoles. There was a systolic murmur at the apical and aortic areas. The thyroid was enlarged, but there was no bruit. Distention of the jugular vein was more marked on the left than on the right. There was presacral and ankle edema. Chest x-ray showed left ventricular prominence with an elongated and broadened aorta, consistent with hypertensive heart disease. The radioactive iodine uptake was 64%. She was given 9 millicuries of I^{131} October 10, 1957. At this time she was on serpasil for her hypertension. She was seen January 23, 1958 and had no heat or cold intolerance. Her skin was dry and her face full. She exhibited a positive Woltman's sign and her radioactive iodine up-take was 8%. She was put on l-thyroxine. Her B.P. was 220/120. A Rogitine test was negative. On February 16, 1958 her l-thyroxine was increased. She weighed 155 pounds. By April 24, 1958 the patient was able to work again and was feeling well. Her weight was 149 pounds.

She was seen again November 28, 1958. She weighed 142 pounds, had a B.P. of 205/100 and a regular pulse of 70. She had had menses twice in two weeks and was having flushes. She was given preludin for this. Her thyroxine and serpasil were continued and diuril twice weekly was added.

This patient was last seen February 26, 1959 when she complained of dull high chest pain in the morning unrelated to effort. The signs and symptoms associated with her previous heart failure were, however, conspicuously absent.

On re-examination the patient was a somewhat nervous woman of 44. Her fundi showed only arterio-venous nicking. The left jugular vein was slightly distended, the right not at all. She had a kinked right carotid artery and the thyroid isthmus and right lobe were palpable. Her chest was clear. B.P. 240/115, pulse 66 — regular and full. There was a grade ii aortic systolic murmur. There was no edema. Her skin was warm and fine. She was considered euthyroid, but still had hypertensive and climacteric symptoms. The dosage of Diuril and preludin were increased. The serpasil and l-thyroxine were continued as before.

Discussion

How does hyperthyroidism effect the cardiovascular system? First, hyperthyroidism causes an increase in basal metabolism. As a direct result the body's oxygen requirement is increased, as is the necessity for heat loss via the skin. Partially in compensation for these needs and partially as a result of increased thyroid function the cardiac output is increased. The cardiac output is, in fact, increased more than the oxygen consumption. Consequently this increased output is achieved at the cost of a definite increase in the total daily work of the myocardium[1, 2, 12].

Secondly, hyperthyroidism causes a persistent increase in the heart rate. This tachycardia continues during sleep and illustrates that hyperthyroidism, acts as a continuous burden on the heart[1, 2, 8, 12, 13]. Further, the heart shows an excessive reaction to exercise, as compared to the normal heart, by an increased tachycardia[2]. Tachycardia in hyperthyroidism is a compensatory mechanism presumably due partly to the increased cardiac output, and possibly also to neurogenic functions and the effects of thyroxine itself[2]. Two distinct disadvantages accrue as a result of increased heart rate in hyperthyroidism. First, there is an increase in the total energy expenditure of the heart. Secondly, with an increased heart rate the diastolic filling time is shortened. This becomes a problem in two types of patient. In the patient with mitral stenosis the available diastolic filling time may be the decisive factor in the presence or absence of pulmonary edema. Also, in patients with myocardial failure, tolerance to tachycardia is poor because the cardiac muscle recovery period is shortened[2].

Thirdly, abnormal rhythm may occur in hyperthyroidism. The commonest of these is atrial fibrillation which may be either paroxysmal or chronic[1, 2, 6, 7, 10]. This atrial fibrillation is less well controlled by digitalis than in euthyroid patients and frequently disappears as thyroid function returns to normal. It is important in hyperthyroidism because it tends to produce further tachycardia[2].

In hyperthyroidism there is an increased systolic pressure (associated with increased cardiac output) and a decreased diastolic pressure (associated with peripheral vasodilation)[2, 12].

The velocity of blood flow is strikingly increased in hyperthyroidism. This is proportional to the increase in B.M.R. and is associated with the increased cardiac output. This is so constant that an unexpectedly short circulation time (in the absence of beri beri, severe anemia or chronic pulmonary disease with cyanosis) should lead the physician to suspect the presence of hyperthyroidism[1, 2, 12, 13].

Stewart and Evans, intrigued by the fact that despite the common recognition of flushed skin and warm extremities in hyperthyroidism no measurements of peripheral blood flow had been undertaken, proceeded to measure peripheral blood flow in 18 patients with hyperthyroidism. They found that in all 18 the peripheral blood flow increased in hyperthyroidism when the B.M.R. increased, decreased during iodine therapy with a parallel fall in B.M.R. and fell further after subtotal thyroidectomy[14].

How does hyperthyroidism produce heart failure? Increased cardiac work as described above obviously plays a part, but is almost certainly not the only factor[2]. Changes in myocardial metabolism have been suggested as precipitating factors. Perhaps the somatic muscle weakness evident in many patients with hyperthyroidism has a counterpart in the cardiac muscle. There is, however, no clear evidence for this as yet. Another possibility lies in the changes of the labile phosphate supply of myocardial muscle. The reduction of A.T.P. and creatine phosphate in experimental hyperthyroidism is of sufficient magnitude to suggest that limitation of high energy phosphate bond storage, and hence immediately available energy for muscular contraction, may be an important factor in reducing cardiac work capacity[15].

Thiamine deficiency may also play a part, as it is well established that increased caloric expenditure increases the thiamine requirement, and evidence of thiamine deficiency has been found in patients with hyperthyroidism[2, 7, 15].

The diagnosis of hyperthyroidism in the presence of heart failure may be obvious or extremely difficult. The cardinal symptoms of hyperthyroidism are:

1) Exophthalmos
2) Thyroid enlargement
3) Nervousness
4) Palpitation

When exophthalmos and thyroid enlargement are absent the other symptoms become more important and therefore one must constantly search for these features[1].

One of the difficulties ensues from the fact that thyrocardiacs have complaints identified with or masked by those of congestive heart failure: dyspnea, palpitation, weakness, chest pain, cough, edema[1, 6, 7]. Consequently a high index of suspicion is important in cases with cardiac failure. This is evident when one considers that there are only a few diseases in which congestive heart failure is common. They are:

1) Terminal picture in rheumatic heart disease
2) Commonly in cardiovascular syphilis
3) Heart changes associated with arteriosclerosis
4) In conditions with prolonged hypertension.

One of the few other conditions with which congestive heart failure frequently occurs is hyperthyroidism[6]. Failure to remember this is another reason why hyperthyroidism in congestive heart failure is missed. Some important clues in the diagnosis are: transient atrial fibrillation, especially if it does not respond to digitalis, undue weight loss (especially if it occurs in the presence of a good appetite), nervousness, finger tremor, excessive perspiration, transient glycosuria, transient diarrhea or bowel hyperactivity, weak quadriceps, frequent premature beats and unexplained tachycardia[1, 8].

Another problem in diagnosis stems from the similarity of signs and symptoms of hyperthyroidism to those of mitral stenosis. Atrial fibrillation, hyperactive heart sounds with an accentuated first sound, a diffuse snapping apex beat, and hyperactive cardiac contraction on fluoroscopy with prominence of the left mid-cardiac segment are common to both disease processes[1, 7, 16]. In cases of uncomplicated hyperthyroidism, Greenberg et al found prominence of the left mid-cardiac segment to be present in 67% of cases[16]. A systolic thrill and murmur are frequently found in hyperthyroidism and with a fast heart rate they may be taken as presystolic.

A much emphasized point in the diagnosis of hyperthyroidism in the presence of heart failure is that the standard methods of treatment of congestive heart failure are of little or no avail. The patient continues to have edema, dyspnea and atrial fibrillation despite complete bed rest and digitalis and diuretics in amounts usually adequate to abolish congestive failure. Why this should occur in thyrocardiacs is not clear. It has been suggested that digitalis and thyroxine may be synergistic in their action and cause injury to the heart muscle[2]. This has not been well authenticated as yet, but, whatever the cause, patients refractory to the standard methods of treatment of congestive heart failure must be investigated for hyperthyroidism.

Once the presence of hyperthyroidism is suspected in a patient with congestive failure, how do you confirm the diagnosis? The basal metabolic rate (B.M.R.), previously the only test of thyroid function, is still useful where others are not available, but may give confusing results for two reasons. First, in almost 50% of euthyroid patients the B.M.R. is in the pathological hyperthyroid range[17]. Secondly, the B.M.R. is elevated in congestive failure and values of as high as +30% in the presence of congestive failure could result from either hyperthyroidism or congestive failure[1]. However, in defence of the test, a low B.M.R. does rule out hyperthyroidism because a well performed B.M.R. does not give false negative values.

The protein bound iodine (P.B.I.) is technically a difficult test to perform, but in a good laboratory, a valuable one, for it reflects a high degree of correlation with thyroid function. A P.B.I. of more than 7.5-8.0 ugm% indicates hyperthyroidism.

The radioactive iodine uptake is another test of thyroid function in common use. When 60% or more of a calculated dose of I^{131} is picked up by the thyroid in 24 hours the patient has hyperthyroidism[1]. The upper limit of normal is 45-50%[17]. This test is also excellent, but is invalidated by previous intake of iodine as food, medication or for radiography before the test is performed.

Kaplan prefers to use a profile of tests to diagnose thyroid function in order to obtain a more accurate assessment of thyroid function, but feels if only one test is to be used as a screening test, the P.B.I. is the estimation of choice[17].

Our three cases have illustrated many of the features usually found in hyperthyroidism with associated heart failure. All three were women which follows the general findings of a marked predominance of females in cases of hyperthyroidism and an even more marked predominance in those who go into heart failure as a result of hyperthyroidism[7]. All three had dyspnea, palpitations, ankle edema and tachycardia. Two of three had atrial fibrillation at one time or another during supervision. All three had evidence of some heart disease. One had hypertensive heart disease, one had arteriosclerotic heart disease and the third had evidence of both arteriosclerotic and rheumatic heart disease. All three were missed as cases of hyperthyroidism when first seen and all three have responded well to treatment of their thyroid disease whereas they had been refractory to treatment of their heart disease alone.

The means of diagnosing hyperthyroidism in the presence of congestive heart failure and the importance of making this diagnosis have been emphasized in this paper. The reasons for this are twofold. First, it is a diagnosis frequently missed despite the attention given this problem in the literature. Secondly, the hyperthyroidism can be cured by appropriate treatment. If it be the sole cause of congestive heart failure, this also is cured. In fact, it is apparently the only cause of congestive heart failure which is completely reversible by the appropriate treatment[7]. Even if it is associated with other heart diseases, the degree and duration of relief obtained is often remarkable.

Treatment

The methods of treatment of hyperthyroidism masked by heart failure are directed at both the thyroid disease and the heart disease. First, regarding the heart failure, the usual methods of treatment are usually necessary and include bed rest, diuretics and digitalis. Where hypertensive or rheumatic or arteriosclerotic heart disease coexists, measures appropriate to the therapy of these diseases may be included in the regimen.

In those patients in whom no other factor than hyperthyroidism can be implicated in the etiology of heart failure, antithyroid treatment alone may completely relieve the heart failure. In view of this factor and of the finding that thyrotoxicosis alone is a not infrequent cause of heart failure[7] and also in view of the previously stated possibility of the danger of the use of digitalis in hyperthyroidism[2] the importance of diagnosing hyperthyroidism in the presence of, or as the cause of, heart failure becomes obvious.

Secondly, therapy directed toward the thyroid gland should be instituted as soon as it is diagnosed as a part of the problem. There is no need to wait until the failure is well controlled before beginning antithyroid treatment. Hamilton had shown as long ago as 1924 that patients thought to be very poor risks for surgery were dramatically improved by subtotal thyroidectomy, and with a remarkably low mortality rate[6]. This was substantiated by Likoff and Levine in 1942, who found a lower mortality in the older group of thyrocardiacs than in the younger non-cardiac group of patients with exophthalmic goitre alone[7].

As to the methods of antithyroid treatment, there seems to be general acceptance of surgery or radioactive iodine therapy as the methods of choice. Antithyroid drugs have proven less satisfactory because of the length of time required for treatment and the toxic complications frequently encountered.

Surgery until recently enjoyed undisputed popularity as the method of choice because it could be performed after rapid and relatively precise medical preparation and because the results were good and the operation safe. Recently, however, the use of radioactive iodine has become widespread in hyperthyroidism and the results are such that this method of treatment must be considered as equal in rank to surgery as the treatment of choice[18].

Once treatment of the thyroid disease has been instituted, it is then necessary to follow the patients for years, supervising their state of health. This is necessary because in both surgery and in the use of I^{131} exactness is difficult to achieve and only supervision over a period of time will ensure the adequacy of treatment. In those cases where therapy has been either excessive or insufficient appropriate steps may then be instituted.

The three cases presented in this paper were all treated with I^{131}. In one patient, one treatment with I^{131} was sufficient to produce a euthyroid state. Another patient required two treatments of I^{131} before a sufficient decrease of hyperthyroidism was obtained. The third patient after one dose of I^{131} was slightly hypothyroid and has required l-thyroxine up to the present time. At the time of writing, all three patients are euthyroid, have no evidence of congestive heart failure and are subjectively feeling much better.

Summary and Conclusions

1. The problem of hyperthyroidism masked by congestive heart failure has been presented and discussed from the points of view of the mechanism of production of failure in hyperthyroidism, the problems and clues in diagnosis, the importance of diagnosis in terms of treatment and prognosis, and the methods of treatment.

2. Three cases of masked hyperthyroidism in whom the diagnosis had been prevously missed have been presented as an illustration of the problems mentioned above.

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Pediatrics

The "Emotional Development" Problem in Pediatric Patients

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The purpose of this article is to present a theory of emotional development in children in a condensed version. The ideas have been gleaned from articles, presentations, and experience in pediatric practice in St. Boniface. It has been found that emotional problems are common, time is often limited, and a preconceived method of effectively handling the minor situations is necessary. Help from the psychiatrist must be sought for the more complex, but every doctor should be prepared to aid parents and children with many psychological distresses without resorting to the answer: "He will grow out of it."

The mind or personality of an individual may be described as having three compartments: one for the animal drives or urges, one for the subconscious memory of "don'ts," and one for the conscious control. The first, you are born with; the second, you learn; and the third is reasoned out by intellect. Emotional maturation consists of full development of the subconscious memory of "don'ts." What is a normal personality? One which responds happily to most situations; satisfying the drives without strain to the intellect and without conflict with social laws. This allows full use of the intellect without undue emotional stresses reducing efficiency.

A newborn baby is a complete and separate individual, who at the same time is dependent by physical necessity. He is not born emotionally dependent. His drives are to be warm, comfortable, and have his hunger satisfied. If his drives are not satisfied, he cries. There is no control. From here personality develops in a series of stages. Each stage demonstrates its own characteristics, so that a child's development may be measured by the application of these stages. Each move is not made all at once, but more like a baseball player, stealing a base. A great many exploratory tries are made before the final break for the next base. This is because of "separation fears" which account for much of the unusual behaviour in children.

Stage I:

Age: 2 - 6 months. The infant learns to love mother. Because comfort and hunger urges are satisfied, the baby becomes emotionally dependent, or in love with the one who loves him. Crying may represent not only pain, but a desire to be close to the loved one.

Stage II:

Age: 6 - 18 months. In learning to crawl and walk the infant learns of his physical independence or individuality. He will experience his first separation fears and demonstrate these by reverting to complete emotional dependence upon mother. The periods of comforting will lessen and he will be satisfied finally in his new position, as an individual able to move about freely.

Stage III:

Age 1½ - 2 years. The demonstration to his mother that he is truly a separate person, is during the automatic "no" stage. This begins with saying or doing the opposite as directed and progresses to deciding entirely separate enterprises.

Stage IV:

Age: 2 - 3 years. The child demonstrates his physical independence by wandering and exploring. This stage is attendant to exaggerated separation fears when he will not move from his mother's side, either night or day. Then suddenly he will take off and become lost.

Stage V:

Age: 3 - 4 years. Complete separation both physically and emotionally is accomplished and the child has his own "body image." This makes him capable of playing with other individuals. His fears are mostly of bodily injury, coupled with an interest in his own body. Fears are common problems, as the child expresses fears of the dark, and of animals and of injury in dreams.

Stage VI:

Age: 4 - 6 years. This is the stage of family relationships. The child is interested in how each

person feels towards the other. Experimenting with professed affection, the child learns of love and jealousy. He is engrossed in how persons in "fairy stories" solve their problems. Fears take the form of fears of people such as robbers, enemies and Indians.

Stage VII:

Age: 6 - 12 years. The child learns to adjust to society outside the family, by applying the same system of progress as used previously:

1. Searching for loving friendship.
2. Individualization by physical prowess or bravery.
3. Individualization by expressing will.
4. Search for freedom and desire to wander.
5. Complete individual but exaggerated anxieties.
6. Experimentation in relationships with people.

Stage VIII:

Adolescence: The pattern of development is re-applied on the background of physical maturation. Just as the infant seeks comfort, the teen-ager has separation fears and requires to touch back to the security of parental love and understanding. At times he will behave in a most immature manner and at others will have confidence to attempt a most adult endeavour.

As a rule, if the initial stages in early childhood have been successful in a normal "loving" way, the development stages through family, society and adolescence will be less hazardous.

What do parents provide?

1. Physical comforts
2. Discipline
3. Love
4. Encouragement
5. Appreciation.

All this is based on a profound interest in the child as an individual and not as an extension of one's own self. Parents, who have had a normal childhood development themselves and are well-adjusted in marriage, rarely have problems with their children.

A baby is born with a chemical and nervous system which has been flavored not only by heredity, but also by the mother's state during pregnancy. Hence parents who are quite tense and excitable, reacting very quickly to stimuli, may naturally have a baby born potentially with the same qualities. The way children react, even from early infancy, to their basic instincts or drives varies considerably.

In actual practice 80% of complaints are either training problems, such as toilet training, sleeping habits, or superficial emotional problems which are not too difficult to help. 20% are more serious such as "brain injured" children, or serious emotional disturbances to anxiety, inferiority or rejection.

Recognition of the symptoms of emotional disorders is more valuable when the situation has not progressed to a serious extent. General practitioners will be able to advise parents who in themselves are not adjusted. In pediatrics you look for the "soft-signs" which mean that a child is emotionally immature. He is performing at a stage below his chronological age.

A great many awkward complaints are posed to the doctor, which come under the broad category of "behaviour problems." Examples are excessive crying, enuresis, crying at night, nervousness, abdominal pains, nervous tics, breath - holding, temper-tantrums, nail-biting. A great many other complaints come under the term psychosomatic. If a diligent search fails to reveal an organic cause, then what should the doctor do?

If the child is young, assess the child's and the parents' intelligence and have the parents return for an interview alone. At this time attention should be directed firstly to the parents' emotional state. Next, a lack of knowledge of normal development may have led to serious misunderstandings. Re-assurance in this department will often solve the problem. The parent-child relationships with the other members of the family should be discussed.

Frequent situations encountered:

1. Failure to supply discipline — the knowledge necessary for the memory of don'ts.
2. Failure to stimulate the child's ego with appreciation and encouragement.
3. Lack of time and interest for a sensitive child because of other children or both parents away at work.

In the case of an older child, it would seem best to interview the child alone first. Rarely will direct questions get positive results, but it is most important to decide how much self-confidence he has.

Frequently the situation resolves itself into an immature personality in the child, with vague non-supportive parent-child relationships.

The parents must be convinced that, for instance, the enuresis is due to the child's lack of self-confidence. From there, they will admit the benefit of psychological advice.

For the sake of simplicity and brevity, four raw materials for the child's personality development are discussed briefly:

1. Love — the apparent variety.
2. Discipline — forever consistent.
3. Interest — patient and understanding.
4. Praise — to build the self-confidence.

Studies in juvenile delinquency suggest that parent-child relationships in early childhood may be the important factor. Certainly self-discipline is based on self-confidence and the background of training discipline in young children.

Doctors should be prepared to recognize emotional immaturity and approach the problem in a positive manner.

Anesthesiology

Controlled Hypothermia and Its Use in Severe Injuries

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Introduction

During Napoleon's retreat from Moscow in 1812, one of the surgeons, Baron Lavrey, noticed that the soldiers who had their legs amputated after exposure to the severe cold and before their legs rewarmed, suffered no pain. Later on (1937) F. M. Allen[1] advocated this type of analgesia for operations on the limbs after chilling, but not freezing the tissues. This proved to be very successful and is still being used today under certain circumstances. Other workers, spurred on by curiosity and observation of hibernating animals, have long been experimenting with the cooling of the whole organism in animals and in man, in order to determine the benefits that might be obtained by this cooling. Fay[2] in 1938 described his cryotherapy for the relief of pain and treatment of malignancies. He first treated malignancies with local cooling, but later cooled the whole body for treatment of cancer in regions not accessible to local cooling. Some patients were kept cooled for up to eight days at temperatures ranging between 25 - 32 degrees C.

In 1950, Bigelow[3] and others, working on dogs, showed a correlation between the body temperature and oxygen consumption. They found that at 30 degrees C, the oxygen consumption in these dogs was reduced by approximately 50% and at 25 degrees C, the reduction was 65%. Under these conditions, it was concluded that the time that the brain or other organs can survive, if deprived, either wholly or in part, of its oxygen supply, will be considerably increased. In the same year Laborit and his co-workers[4] introduced their "lytic cocktail" where body cooling was produced and aided by using a mixture of drugs. For some time now, hypothermia has been used as an aid in heart surgery, neurosurgery and cardio-vascular surgery and as a part of the treatment in severe injuries. Postel, Reid and Hinton[5], in 1957, published a paper about the use of hypothermia in experimental hemorrhagic shock in dogs. From their results, it was concluded that the clinical use of hypothermia in the therapy of certain shock states was warranted.

Methods of Producing Hypothermia[6]

Before starting the cooling process, the important protective reflexes must be controlled — shivering and vasoconstriction.

If vasoconstriction is permitted, there will be a delay in the fall of body temperature and there is danger of frostbite.

These two reflexes can be controlled in a number of ways:

1. By deep anaesthesia, e.g.: by ether.
2. By light anaesthesia, combined with the use of muscle relaxants and vaso-dilator drugs.
3. By the use of the "lytic cocktail"[4] which, with its important phenothiazine drugs, will control the temperature regulating system.

Once control of these reflexes is established, cooling may be produced by a variety of methods.

A. Surface Cooling

This is an effective and simple method for cooling the body. After the patient is anaesthetized, or sedated by the "lytic cocktail," ice bags are packed all over the body to cool the body surface. Cooling is produced by direct conduction through the tissues and by the blood circulating through the cooled parts. This method is slower, but has the advantage of simplicity. Other methods of surface cooling include blowing air (by electric fans) over the body, kept wet with cold water; wrapping the patient in a cooling blanket or by immersing the patient in a bath of cold water.

Cooling should stop when the body temperature (measured rectally or in the oesophagus) has reached within 3 - 4 degrees C of the desired temperature as there is always an "after-drop" due to blood circulating through the colder superficial tissues.

B. Body Cavity Cooling

There are two common methods of doing this. The first consists of pouring cool saline solution into the open thoracic cavity. However, it is a very slow process and requires large volumes of saline. Re-warming may be produced in a similar way by using warm saline.

The second method involves the use of a thin rubber tube leading to one end of the balloon and a second tube coming back from the other end. The balloon is introduced into the stomach and then cold water is circulated through the tubes and balloon. Due to the close proximity of the stomach to the liver, heart and spleen, the blood from these organs is cooled first and then circulated to the rest of the body. This also is a very slow method of cooling and is not often used.

C. Extra-corporeal Cooling

Here again two methods are available. The first consists of cannulating an artery (e.g.: the femoral) from which blood circulates through tubing immersed in a cooling solution and is returned to the body via a vein, e.g.: the jugular vein. One major disadvantage is that an artificial arterio-venous shunt is created and this throws extra work on the heart. This could be dangerous in a patient with an already damaged myocardium.

The second method consists of cannulating a vein from which a mechanical pump draws blood and circulates it through tubes in cooling solution and back to another vein. Brock[7], in 1956, modified this technique by inserting a cannula into the right auricular appendage and passing it into the superior and inferior vena cavae. Brock states that in order to avoid a too rapid cooling of the heart, the body temperature should fall one degree C each five minutes. Some workers have been using extra-corporeal cooling to selectively cool the brain without cooling the rest of the body and have produced good results in work on dogs[8].

The chief aim in the use of hyperthermia is to reduce the metabolism of the animal or patient and in so doing to reduce the oxygen requirements. Bigelow and others (1950) showed in dogs, that the oxygen uptake of the tissues is reduced by about 50% at 30 degrees C and that with a further fall of temperature to 25 degrees C, the reduction of oxygen uptake is 65%.

At low temperatures, the dissociation curve for oxyhaemoglobin is shifted to the left, i.e.: there is an interference with the release of oxygen. The question therefore arises if the reduction in oxygen uptake is due to this factor, or to decreased need by the tissues for oxygen. Echenhoff does state that oxygenation of the tissues is adequate[6].

The reduced need for oxygen by the brain under hypothermia has been shown by McMurney and his colleagues[9]. They showed that in normothermia monkeys subjected to complete cerebral anoxia cessation of E.E.G. activity occurred after one minute, but that when the body temperature was reduced to 26- 23 degrees C the E.E.G. activity continued for fifteen minutes.

Another consideration is the change in oxygen consumption of different tissues. At 26 degrees C the oxygen uptake of the whole body is about 40% normal but at that same temperature that of the heart is only 50%. It has been shown that the cerebral uptake showed little change until 31 degrees C was reached and then it dropped sharply to about 75% of normal by the time the temperature reaches 27 degrees C. Further cooling reduces the oxygen uptake further, but more slowly[10].

Therefore the optimum temperature seems to lie between 27 - 31 degrees C. Temperatures below 27 degrees C are dangerous because of the high incidence of ventricular fibrillation.

Some workers claim that myocardial hypoxia is the important factor for the production of ventricular fibrillation and that this in turn is probably due to the shifting of the dissociation curve for oxyhaemoglobin to the left.

Another important consideration is that of the pH of the blood. Changes in pH can cause cardiac arrhythmias and the combination of pH change and myocardial hypoxia is particularly dangerous.

With cooling there is an increased viscosity of the blood and an alteration in the coagulation time. After prolonged cooling the platelets diminish and the clotting time increases. This however, has not been the cause of intractable haemorrhage, except when there has been some associated clotting deficiency as occurs with cirrhosis of the liver.

During hypothermia, there may be a fall in blood pressure, due to a fall in cardiac output and vasodilatation. The drop in cardiac output and vasodilatation results from the low temperature and/or from the concomitant use of drugs such as chlorpromazine. This fall in blood pressure is not usually serious except when there exists a severe myocardial insufficiency.

If surface cooling is used, salt should not be added to the ice packs or ice bath as frostbite may be produced. Occasionally, patients who have had surface cooling, develop areas of scleraderma and burns. Pressure points during hypothermia must be carefully observed and padded, as tissue necrosis occurs easily following prolonged pressure.

The metabolism is, of course, affected by hypothermia. A response similar to the reaction to stress is seen after prolonged cooling particularly when the surface method is being used, and the administration of cortisone during hypothermia has been recommended.

Hyperglycaemia may occur during cooling. During the cold state glucose is metabolized slowly and if I.V. drips of glucose are being given there may be a marked rise in blood sugar. In these cases, therefore, too much I.V. fluids should be avoided, and it is suggested that a 2.5% solution of glucose should be used instead of the usual 5%.

During prolonged cooling careful attention must be paid to serum electrolytes. Blood lost in a severe injury and into tissues during a fracture should be replaced.

Hypothermia is used in surgery as mentioned above, but it may also be used to treat severe head injuries, particularly where oedema of the brain leads to or aggravates brain cell anoxia, accident cases with multiple injuries and severe blood loss, some shock states, and after successful cardiac resuscitation.

Presentation of Cases

At the St. Boniface Hospital the department of anaesthesia is put in charge of instituting and maintaining "hypothermia" whenever the attending physician and the anaesthetist feel that it is indicated. The technique used is as follows: The "Lytic Cocktail" of Laborit[4] which consists of Meperidine 100 mgms., Promethazine 50 mgms. and Chlorpromazine 50 mgms. is placed into 500 or 1000 cc of 5% dextrose and water and infused through a vein either by needle, catheter or cut-down. Ice bags are placed about the patient's body after the skin has been protected with a layer of vaseline; this has been found necessary in order to protect the skin from burns, and has been found

not to interfere with the production of hypothermia. The temperature is monitored with the use of a Tele-Thermometer and a rectal probe. The rate of the infusion is titred to avoid shivering, rigidity or spasticity.

Case 1

Mr. J. G., Hospital No B-3271, age 41, August 24th, 1958: Admitted to the hospital following a car accident. Patient was unconscious and immediate investigation revealed intracerebral and subarachnoid hemorrhages, and multiple rib fractures. Hypothermia was decided upon and using the "lytic cocktail" and ice packs, he was cooled to a rectal temperature of 29-30 degrees C. On August 26th, he vomited and aspirated stomach contents after which he developed a post-atelectatic pneumonia. However he did improve, and hypothermia was discontinued on August 31st. He continued to improve and was discharged on October 11th, 1958. At the present time he is still at home. His physical status is good but he is unable to carry on at any job. His relatives maintain that there has been a marked change in his personality since the accident.

Case 2

Miss H. D., Hospital No. B-281, age 18, Jan. 4th, 1957: Admitted to the hospital following a car accident. Patient was unconscious with intermittent periods of severe cerebral irritability. She was extremely restless, spastic and hyperexia developed. Hypothermia was started with the "cocktail" and ice-packs. The temperature was maintained between 31 -33 degrees C. On January 29th, she developed a massive Ludwig's angina and sub-hyoid infection which was incised on February 1st, under hypothermic anaesthesia, but her condition continued to deteriorate and she died on February 3rd. Post-mortem revealed cerebral oedema, lobar pneumonia and cellulitis of the neck.

Case 3

Mr. R. C., Hospital No. B-14128 age 26 Aug. 1st, 1958: Admitted to the hospital following a car accident. Patient was not unconscious but was complaining of progressive blindness since the accident. X-rays of the skull revealed multiple fractures. On August 8th, he was taken to the O.R. and several bone fragments removed. Sept. 3rd, he fell out of bed and became unconscious. An immediate craniotomy revealed bleeding from the site of the previous operation. There was a great deal of swelling, so much in fact that part of the frontal lobes had to be resected before the bone flaps could be replaced. In an effort to reduce the cerebral oedema hypothermia was instituted postoperatively. The "lytic cocktail" and ice packs were used and the temperature kept between 32 -33 degrees C. His condition did not improve and he eventually died on Sept. 28th. Post mortem showed bilateral broncho-pneumonia and abscesses of the remaining positions of both frontal lobes.

Case 4

Mr. J. W., Hospital No. B-17733, Age 13, Sept. 26th, 1958: Admitted to the hospital after being kicked in the head by a horse. The patient was unconscious and extremely restless. Burr holes and ventricular taps revealed intra-ventricular bleeding. Hypothermia was instituted and the temperature brought down to 32 - 34 degrees C. Sept. 28th, the patient died. Post mortem findings were severe cerebral oedema, and contusions, and broncho-pneumonia.

Case 5

Miss M. M., Hospital No. B-18899, age 20, Nov. 17th, 1957: Admitted to the hospital following a car accident. Patient was unconscious, spastic and convulsing at times. X-rays revealed bilateral fractures of the femurs and skull fractures. Temperature rose to 103°F. Hypothermia down to 32 degrees C was instituted and maintained around this temperature for about three weeks. Intermittent convulsions and periods of decerebrate rigidity occurred until Dec. 4th, but improvement occurred after this. Dec. 16th both fractures were nailed. Feb. 4th a cranioplasty was done. Feb. 13th she had her teeth extracted and she was discharged on Feb. 20th. Today she is married and all her mental tests are normal.

Case 6

Mas. D. H. Hospital No. B-36024, age 14, Nov. 4th, 1958. Patient was admitted to the hospital after sustaining a gun-shot wound to the left thigh. The femoral artery had been sectioned by the bullet and the patient was in severe shock due to extensive blood loss. The boy's parents were Jehovah Witnesses and refused to give permission for blood to be used. After 36 hours his condition became critical, the haemoglobin had dropped to slightly below 4 grams, his temperature rose and he became extremely restless. Hypothermia was then instituted in an effort to reduce his oxygen requirements. The temperature was lowered to 32 degrees C and maintained at this level for 7 days. After this period of time his condition had not materially improved. It seemed that he might be allowed to receive blood transfusions and he was allowed to warm up in order to assess his condition more accurately. However he died during this period of rewarming. Post-mortem findings were cerebral oedema, marked anemia of all the tissues and broncho-pneumonia.

Case 7

Mas. M. K., Hospital No. 59-3822, age 7, March 1st, 1959: Patient was admitted to the hospital following car accident. Patient was unconscious, decerebrate rigidity and convulsions were present and the temperature began to rise to 102°F. On March 3rd, hypothermia was begun and the temperature was reduced to 33 degrees C. The temperature was maintained between 33 and 34

degrees C. On March 8th hypothermia was discontinued but twitching and convulsions began again and the hypothermia was recommenced. March 17th the application of the ice packs was stopped and the rigidity controlled with small amounts of "lytic cocktail" only. March 22nd the patient's level of consciousness is lighter, he responds to moderate stimulation in a very normal manner and it seems certain that he will recover. Whether he will become completely normal mentally remains to be seen.

The above cases summarize our experiences in the use of hypothermia in severely injured patients. It is, of course, too small a series from which to draw any conclusions; however certain aspects of these cases and the treatment should be emphasized. Of the seven cases, Case 5 is alive and well, Case 1 is alive and not normal mentally, however he may improve with the passage of time; Case 7 is alive, improving, and may resume a normal life. Cases 2, 3, 4 and 6 died; the most important features at post mortem were severe infections such as broncho-pneumonia, other abscess sites, and cerebral oedema. From these cases it seems apparent that the hypothermia did not produce any reduction in cerebral oedema, either that or that the brain damage was so great that no agent or technique could possibly do any good. Of those that recovered, i.e., Cases 1, 5 and 7, a common factor was decerebrate rigidity, convulsions and spasticity.

Maciver and his co-workers[11] discussed treatment of severe head injuries and noted that the best prognosis attended these cases in which the patients had hyperpyrexia with tonic fits superimposed on decerebrate rigidity (our cases 1, 5 and 7). They stressed particularly the use of triple plasma for the relief or prevention of secondary edema, and the use of antibiotics to combat infection. From our experience it is doubtful whether hypothermia reduced any existing cerebral edema; earlier institution of this technique might however have prevented the occurrence of edema.

Case 6 differed from the other cases in that direct trauma to the brain was not a factor. Ferguson, et al[12] in March 1958, bled three groups of dogs to 35% of their measured blood volumes. Group I, normothermic dogs were bled to the desired level, all of these survived. Group II, the dogs were cooled to 25 degrees C before bleeding, 80% died; Group III, these dogs were bled while normothermic and then cooled to 25 degrees C, all survived. The suggested value of hypothermia as a method of treatment of hemorrhagic shock was not confirmed or denied. Other investigators however have shown that hypothermia plays a definite role in increasing the resistance of the organisms to trauma and oxygen lack[5, 13, 14]. We therefore feel that hypothermia should be used in cases of severe hemorrhagic shock, especially when, for some reason, it is impossible to replace the deficit in the blood volume.

Summary

In this presentation the authors have reviewed the history and physiological effects of hypothermia. Seven cases of severely injured patients in whom hypothermia was used as part of the treatment have been presented. Three survived and four died for a survival rate of 43%. The following adjuncts to routine therapy in this type of case is suggested. 1. The use of hypothermia to decrease the oxygen demands of the brain, heart, liver and kidney and to control hyperpyrexia. 2. The use of a lytic cocktail to control rigidity, restlessness, and hyperpyrexia. 3. The use of massive doses of antibiotics to control infection. 4. The use of concentrated albumin to prevent and treat cerebral edema. 5. Early tracheotomy for ease of suction and to improve the ventilation.

The success of this technique depends on an adequately trained nursing staff who will pay meticulous attention to frequent turning of the patient, maintaining of the intravenous, oral and tracheotomy hygiene, protecting the patient from skin necrosis and burns, and avoiding rigidity and restlessness.

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Obstetrics & Gynaecology

Hypertension Associated With the Use of *Ergonovine and Methylergobasine

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Ergot is one of the older drugs of the medical armamentarium and has been used in obstetrics for the past four centuries. The production of chemically pure alkaloids and the synthesis of methylergonovine resulted in a growing employment of the drug. In our present practice ergot alkaloids are routinely used as a prophylactic against haemorrhage in the third stage of labour and early puerperium.

The administration of these drugs is followed sometimes by undesirable vasomotor effects, such as sudden elevation of blood pressure with headache and palpitation. Since the articles dealing with this problem are somewhat contradictory we were stimulated to carry out a clinical survey in order to assess and compare the effects on the blood pressure exerted by these drugs. This investigation made comparison of the effects of ergonovine and methylergobasine.

Method

During the first period ergonovine was administered to 207 patients. In the second period methylergobasine was given to 241 patients. There were 35 controls. A larger number of controls was difficult to obtain since complication of the third stage and of the early puerperium necessitated the administration of oxytocics. The blood pressure was recorded on admission, immediately after the delivery of the infant, at 30 minutes, one hour and 24 hours following the administration of the drug. Both drugs were given intramuscularly.

These drugs were employed under similar clinical conditions. The patients received an analgesic during the first stage of labor, and during the second stage of labor a light general anaesthesia was given. No spinal, caudal or epidural anaesthesia was employed.

Results

Table I shows the mean values of the blood pressure of the unselected cases and of the control group.

TABLE I

Mean value of B.P.	Ergonovine	Methylergobasine	Control
On Admission	122/77		120/76
Immediately after Delivery of the Infant	126/80		123/74
30 Minutes after Administration of Drug	131/83	127/82	121/75
1 Hour after Administration of Drug	129/81	123/78	117/76
24 Hours after Administration of Drug	114/72	115/73	116/74

We investigated the vasomotor effect of the drugs on a hypertensive group of patients. Those patients were admitted into the group who had either a systolic pressure higher than 90 mm Hg. immediately after childbirth. The blood pressure on admission was not considered since a large percentage of patients belonging to this group were admitted several days before the onset of labor and received antihypertensive treatment before and during labor. The mean value of the blood pressure 30 minutes after the administration of the drug, which represented the peak in blood pressure rise, was compared to the mean recorded immediately after the delivery of the infant.

The same problem was investigated in a normotensive group of patients. The criterion for admission into this group was a systolic pressure below 140 mm Hg. and a diastolic less than 90 mm Hg.

Ergonovine was given to 40 hypertensives and 167 normotensive patients. Methylergobasine was given in 55 hypertensive and 186 normotensive patients.

Table II compares the mean values of the blood pressure of the hypertensive and normotensive groups treated by oxytocics and also of the normotensive group of the control series.

Discussion

As mentioned before, the articles dealing with this problem are somewhat contradictory. Cerone, Pannullo, Ferguson and Reid did not observe any vasopressor action when Methergine (Methylergobasine in Canada) was given parenterally. On the other hand Carvalho and associates have found definite hypertensive effect following the administration of any of the oxytocics and question the routine

* Also known as Ergometrine.

TABLE II

MEAN VALUES of B.P.	HYPERTENSIVE Ergonovine	HYPERTENSIVE Methyl-ergobasine	NORMOTENSIVE Ergonovine	NORMOTENSIVE Methyl-ergobasine	Normotensive Control Group
Immediately after Delivery of Infant	149/92	146/94	120/75	119/76	119/72
30 Minutes after Administration of the Drug	147/92	144/89	129/81	123/79	120/74

Table III compares the blood pressure changes at different levels.

TABLE III

B.P.	HYPERTENSIVE		NORMOTENSIVE		Normotensive
	Ergonovine	Methyl-ergobasine	Ergonovine	Methyl-ergobasine	Control Group
Dropped	56.7%	50.9%	23.5%	32.4%	48.3%
Increase less than 10 mm hg.	13.5%	21.8%	34.8%	35.1%	34.5%
Increase 10-20 mm hg.	8.2%	12.7%	20.7%	19.6%	10.4%
Increase 20-30 mm hg.	13.5%	10.9%	8.4%	10.1%	3.4%
Increase 30-40 mm hg.	2.7%	3.7%	7.6%	1.3%	3.4%
Increase more than 40 mm hg.	5.4%	0.0%	5.0%	1.5%	0.0%

employment of the uterotonics. In their series methylergobasine proved to exert the least effect on blood pressure. Schade investigated the hypertensive action of these drugs on normo- and hypertensive group of patients. He has found less vasopressor effect in hypertensives than on normotensives and concludes that oxytocics can be given with comparative safety in case of hypertension. Cartwright did not experience any significant rise in blood pressure or any undesirable side effect when Methergine was given, and, in his opinion, patients with hypertension should not be deprived of oxytocics. McGinty found a significant elevation of blood pressure (20 mm Hg.) in 26% of patients where either ergonovine or methylergobasine was given. Severe elevation of the blood pressure (to more than 170 mm Hg. systolic) was seen in 12% of the ergonovine series, 6% of the Methergine series. Crunden and associates did not see blood pressure rise exceeding 18 mm Hg. in any case following parenteral administration of methylergonovine.

Our series definitely shows that both ergonovine and methylergobazine cause an elevation of the blood pressure. The first table shows that ergonovine has a more pronounced effect than methylergobasine, and this effect seems to be of a longer duration. We think that the number of investigated cases is large enough to attribute significance to the mean value illustrated on Table 1 and 2.

From Table 2 we think it is justifiable to conclude that the hypertensive action of either ergonovine or methylergobasine is less in hypertensive than in normotensive patients.

Table 3 shows that in about half of the cases the blood pressure dropped in the hypertensive group, while the same alteration occurred in about one third of the normotensive group. It seems probable that in certain cases these drugs lower the blood pressure instead of raising it. Ergot alkaloids act on the blood pressure in different ways. They exert a direct vasoconstrictor effect on the vessels. On the other hand the sympatholytic action of ergot alkaloids is well known. The latter effect is probable of central origin. It is such a pronounced property of the dihydro ergot alkaloids that these compounds are used therapeutically as antihypertensives (e.g. Hydergin). It is probable that in the patients with hypertension, when the sympathetic tone of the autonomic nervous system is increased, the sympatholytic effect of these alkaloids prevails over the direct vasoconstricting effect.

The above facts and considerations should not influence us into a false sense of safety in administering ergot preparations to hypertensive women. In this series 8.1% of the patients receiving ergonovine and 3.7% of the patients receiving methylergobasine the systolic pressure rose more than 30 mm Hg. in the hypertensive group. In the normotensive group 12.6% of the patients showed an elevation of more than 30 mm Hg. of the systolic pressure following the injection of ergonovine. This sudden rise in blood pressure may provoke eclampsia in toxaemic patients and may be disastrous for a patient with cerebrovascular disease.

Conclusion

1. Both ergonovine and methylergobasine tend to elevate blood pressure. The hypertensive effect of ergonovine is more pronounced than of methylergobasine.

2. Normotensives respond more often with sudden elevation of the blood pressure than hypertensives This fact does not mean security when ergot preparation are given for hypertensive patients, since in a certain number of cases sudden rise may occur, threatening the patient with eclampsia or cerebrovascular accident.

3. We think that the routine employment of these oxytocics should be taken into consideration. We do not recommend the prophylactic administration of ergot preparations in cases of severe hypertension. On the other hand complications of the third stage and early puerperium justify the use of oxytocics.

4. Methylergobasine seems to be preferable to ergonovine especially in cases of hypertension.

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Radiotherapy

On the Uses of Cobalt Beam Therapy

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Cobalt beam therapy is essentially a Canadian development—relatively recent in nature—the first units having gone into use in London, Ont. and Saskatoon about eight years ago. The first Manitoba units came into Winnipeg in 1953 and in St. Boniface in 1956.

Radiation from the artificially radioactive cobalt source is to all intents and purposes gamma radiation, similar to that of a supervoltage x-ray machine of one to three million volts energy. Such radiation has some disadvantages and many advantages.

Disadvantages

The radioactive cobalt source has a half life of five years, i.e.: the intensity of radiation energy in the source decreases by 50% every five years. The source, in fact, has to be renewed every two to three years, if the output in roentgens per minute is to be maintained at an economical level. The source is continually losing strength whether or no the machine is being used. There is a small but definite penumbra around the main beam, i.e.: an area outside of the intended field where amounts of up to 20% of the central dose may be given to surrounding tissue.

Unlike x-rays in the 250 to 500 k.v. range, vital structures within the treatment field such as eyes, etc. cannot be protected by lead shielding. It would take approximately ½" of lead to reduce the incident dose by even 50% and the weight of lead necessary for complete protection would be insupportable.

The cobalt beam is by no means a complete supplanter of the more conventional methods of x-ray therapy. It is of little use in the treatment of skin lesions and unnecessary in the treatment of other lesions near the surface.

Advantages

The advantages of cobalt beam therapy are many, both to radiotherapist and to patient. Unlike x-rays up to the 500 k.v. range, skin reactions are kept to a minimum. Due to the high energy of the gamma radiation, backscatter from the sulphur content in the Malpighian layer of the skin does not occur and the radiation has a point of maximum dosage actually about 0.5 cms. below the skin surface. Another advantage is that whereas conventional x-rays are absorbed twice as much by bone as by soft tissue (which gives any tumor immediately behind bone a definite protection against irradiation) the absorption of the cobalt beam is the same in bone as it is in soft tissue.

The percentage dose at a given depth for any given field size is much greater for the cobalt beam than for the conventional x-ray beam. For instance, with a field size of 15 x 10 cms., a beam of x-rays at 250 k.v., the skin dose of which is taken as 100%, is reduced to 37% at 10 cms. below the skin surface and to 19% at 15 cms. The corresponding percentage depth doses for a 15 x 10 cms. field on the cobalt beam at 10 cms. and 15 cms. are 57% and 40%.

The cobalt beam itself, due largely to its high energy does not have the same "scatter" as an x-ray beam. "Scatter" is a technical name given to the method of absorption of radiation. X-rays have scatter in all directions — forward, side and back. Side scatter is the main phenomenon in x-ray absorption and so the path of an x-ray beam in tissue may be likened to the shape of an isosceles triangle. The cobalt beam absorption, however, is mainly by forward scatter so that the path of the cobalt beam in tissue is rather like a solid straight line.

A further advantage is that patients do undoubtedly tolerate from the cobalt beam large doses of radiation, even to areas known to be easily affected adversely such as the abdomen and the pelvis. It has long been a drawback of conventional x-ray therapy that large doses to these areas are not usually obtained due to the patient's bodily reaction.

Cobalt beam therapy may be used in three ways:

(1) For cure — as the sole method.
(2) For cure — as either a pre or post operative measure.
(3) For palliation only.

Under the first heading may be grouped carcinomata of the following sites: oesophagus, lung, rectum, bladder, larynx, tonsil, fauces, palate, floor of mouth, buccal aspect of cheek, penis. Some bony tumors such as reticulum cell sarcoma and Ewing's Tumor should probably be included in this group.

The first three sites are mainly surgical problems, but in cases unsuitable for surgery because of age, associated conditions or lack of the proper appreciation of the wonders of modern surgery, cobalt beam therapy can and should be used as a cure. Ivan Smith of London, Ont. has reported four five-year cures of carcinoma of the oesophagus. I. G. Williams of London has reported several cases of cure of carcinoma of rectum by supervoltage therapy and there is no reason why his results should not be obtained by cobalt beam therapy.

Bladder tumors too large to implant but otherwise operable can easily be treated by cobalt beam therapy. High grade bladder tumors should probably not be operated upon and should have cobalt beam therapy.

Carcinoma of tonsil, fauces and palate, of floor of mouth and of buccal aspect of cheek are all in areas difficult to control by surgery without gross mutilation. Cobalt beam therapy is an excellent method of treating carcinoma in these sites. Some side effects from treatment there may be—dryness of mouth is one — but surely a dry mouth is preferable to a moist area lacking any of the more easily identifiable features of a mouth. Bone damage is rare unless retained tooth roots or carious teeth are present at the time of treatment. Clearance of dental sepsis is essential before treatment.

Carcinoma of penis is eminently curable by the cobalt beam provided the lesion has not invaded the urethra or the corpora. These conditions being granted, there would seem to be little justification for using surgery, as the primary method of control. Admittedly, partial amputation in an early case would almost always be curative but at a price usually esteemed somewhat high. Lymph node secondaries from any of the above diseases, however, should be treated by surgery.

The body tumors which are extremely radiosensitive, such as reticulum cell sarcoma, etc., if solitary, should be treated by cobalt beam therapy. Cure is possible without the mutilation of amputation. As explained above, by cobalt beam therapy as compared with conventional x-ray therapy, bone damage is not likely to occur as a result of radiation.

Pre or Post Operative Therapy

Cobalt beam therapy may be used in combination with surgery in either a pre-operative or a post-operative role in several conditions, viz.: malignant tumors of breast, kidney, antrum, brain, testis and ovary. Its use in the treatment of breast tumors would be in destroying malignancy in the axillary and supraclavicular regions and in the parasternal nodes.

In the post-operative treatment of carcinoma of kidney, its high dose at a depth is useful in delivering a tumor dose to the kidney bed and regional lymph node areas.

In carcinoma of the antrum the risk of bony damage by radiation is greatly reduced by the peculiar physical properties of the cobalt beam and it may be used as a sole method of cure.

The treatment of brain tumors does not give particularly good results no matter what method is employed, but at least with cobalt beam therapy the bone barrier is easily overcome and a known, evenly distributed dose can be given. Post-operative treatment in carcinoma of testis can be effectively given to the iliac and para-aortic lymph node areas with little or no reaction and to a dosage higher than could be achieved by conventional deep x-ray therapy. Similarly treatment of the whole pelvis after operation for carcinoma of the ovary is well tolerated in most cases and again to a higher dose than could be obtained by conventional x-ray therapy. As is well known, cobalt beam therapy is used as an adjunct to intracavitary radium therapy in the treatment of carcinoma of the cervix.

Palliation

Good palliation may be achieved by treating any of the above types of tumors when cure is no longer possible. In many cases where a relatively high dose has to be given to achieve palliation, this could be done only once when using deep x-ray therapy due to its damaging effect on the skin, etc., whereas such treatment can usually be given several times to achieve continued palliation when using cobalt beam therapy. It is also possible now, though not generally appreciated, to give reasonably good palliation to carcinomatous tumors of pancreas, liver and bowel. In fact, cobalt beam irradiation of recurrent rectal carcinoma after failed abdomino-perineal surgery has given extremely good palliative results, even in the presence of tumor actually growing through the perineal wound. Treatment by the cobalt beam produces loss of pain and marked tumor regression despite the relative radioresistance of this type of tumor.

Finally it should definitely be borne in mind that whenever cure is possible by using either cobalt beam therapy or surgery, then cobalt beam therapy should be tried first. If it fails, and it has been known to, then surgery is no less feasible and may even be easier. There is usually no tissue damage and planes of surgical cleavage are not affected. On the other hand, radiation therapy after failed surgery is difficult even with cobalt beam therapy because of the factors known to increase radioresistance which have probably been introduced by the surgical trauma: scarring, poor vascularity, anaerobiosis and sepsis.

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Pathology

Observations on the Histogenesis of Invasive Squamous-Cell Carcinoma of the Uterine Cervix

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It is acknowledged by definition that invasive squamous-cell carcinoma of the uterine cervix is preceded by an anaplastic growth alteration of the cervical mucosa. Whether or not this abnormal intraepithelial lesion, so-called carcinoma in situ or intraepithelial carcinoma, has a determined period of reversibility to normalcy, still remains a matter of considerable controversy among cytomorphologists. Well-documented observations, however, strongly support the view that true epithelial carcinoma in situ, if not treated, will eventually show evidence of stromal invasion[1].

Although the medical literature of the last decade or so is replete with prolific discussions of the treatment of in situ and invasive carcinomas of the cervix, lesser importance has been attached to the mucosal alterations which may, under sustained carcinogenic stimulation, progress to definite intraepithelial carcinoma and subsequently to frank invasive cancer.

An outline of the possible development stages of invasive carcinoma of the uterine cervix will be presented in this paper. Topographic consideration will be given to the histogenesis of carcinoma arising from the squamous epithelium of the ectocervix and to that derived from the endocervix normally invested with a columnar epithelium (Figs. 1, 15, 2, 3). The morphologic features of each distinct entity which may lead to carcinoma will be described and an evaluation of their respective clinical significance will be made.

1. Histogenesis of Invasive Carcinoma of Endocervix

Two theories are currently expressed in an attempt to explain the occurrence of squamous carcinoma in the endocervix. One postulated source of origin is the contiguous substitution of the endocervical mucosa by a progressive and superficially spreading carcinomatous process extending from the neighboring squamous epithelium of the ectocervix. The first postulation, however, does not explain satisfactorily the histogenesis of endocervical squamous carcinoma arising and located distant to the squamous epithelium of the portio. The second and more plausible theory suggests a derivation from the "reserve cells" of the endocervical mucosa.

Reserve Cells

Endocervical reserve cells are probably undifferentiated cellular remnants of the primitive cervical lining. These cells are focally present in most human cervices and are identified as a single subcolumnar layer of well-demarcated cuboidal cellular elements, located in the superficial endocervical mucosa and/or the glandular epithelium. A thin reticulin membrane separates their basal surface from the underlying supporting stroma. Their cytoplasmic mass is devoid of any secretion at this stage of inactivity (Fig. 4).

Exfoliation of such reserve cells is infrequent unless a forceful endocervical aspiration is performed. Fig. 5 reveals a cluster of cellular elements believed to be derived from a patch of reserve cells. Although suggestive of in situ carcinomatous cells to the inexperienced cytologist, they tend to be smaller, more uniform in size, and their nuclear chromatin pattern is not so coarse.

The nature of the activating agents which stimulate the proliferation of reserve cells to mature squamous metaplasia, atypical hyperplasia and intraepithelial cancer, remains obscure. Various factors, however, such as contact traumatism, inflammatory processes and hormonal substances, have all been implicated as responsible mechanisms[2, 3].

Subcolumnar reserve cells in a state of inactivity, when identified in cervical biopsy specimens, probably warrant no further diagnostic or therapeutic procedures, since the greatest proportion obviously remain dormant or develop to squamous metaplasia.

Squamous-cell Metaplasia

Epithelial metaplasia may be defined as an adaptive substitution of a fully differentiated epithelium of one type by a differentiated epithelium of another kind. Within the cervix, squamous-cell metaplasia refers to the replacement of the columnar endocervical surface or glandular mucosae, differentiated toward mucus secretion, by a mature squamous epithelium possessing the morphologic characteristics of normal mucosal squamous epithelium.

The transitional growth pattern of reserve cells to squamous metaplastic epithelium is not always apparent in random sections of the cervix, but the progressive changes may be identified readily in some instances as illustrated in Fig. 6. This modification is represented by an abrupt transformation of a double layer of reserve cells (to the left) to a multilayered metaplastic epithelium (to the right). In the latter there is a continuous maturation from base to surface, the deeper epithelial stratum consisting of small, perpendicular, and uniform reserve

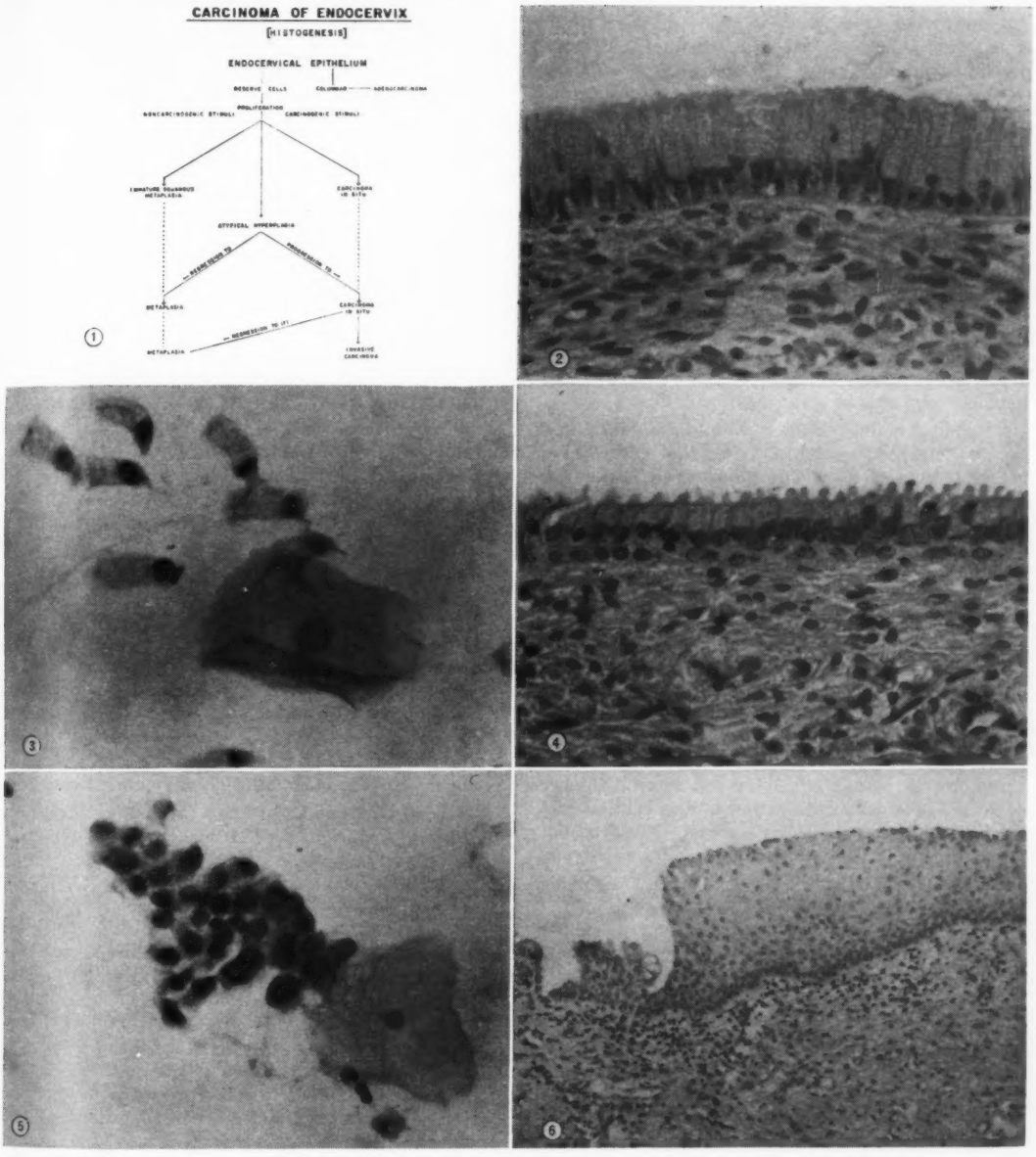


Figure 1. Histogenesis of invasive squamous carcinoma of the endocervix. Note the various developmental stages of reserve cells which may proliferate to squamous metaplasia, atypical hyperplasia and carcinoma in situ.

Figure 2. Endocervical mucosa. The epithelium is composed of tall columnar cells. Reserve cells are not identified in this field.

Figure 3. Endocervical columnar cells exfoliated from the mucosa and identified in spreads.

Figure 4. Reserve cells. Subcolumnar unistratified layer of low cuboidal reserve cells which are distinct from the underlying stroma.

Figure 5. Exfoliated reserve cells. The cellular elements are small, possess a scanty basophilic cytoplasm and contain uniform finely granulated nuclei.

Figure 6. Squamous-cell metaplasia. Abrupt transition from reserve cell nidus to typical multilayered metaplastic epithelium.

cells, while the intermediate and superficial zones are comprised of larger polygonal squamous-like cells. The cellular outlines are distinct and composed of condensed ectoplasm, while the perinuclear endoplasm is focally vacuolated. Maturation is manifested by increased cytoplasmic mass in ratio to nuclear size, absence of nuclear mitoses, and occasionally glycogenation of the superficial cells.

Carmichael and Jeffreson, in their classical description of squamous metaplasia of the cervix, identified such an alteration of the endocervical mucosa in 41 per cent of 400 cervices examined[4], while Auerbach and Pund observed it in 72 per cent of the cases studied[5]. These figures would tend to indicate that the general trend of reserve cell proliferation is stimulated toward varying degrees of squamous metaplasia rather than directed toward dysplastic or anaplastic growth.

Squamous-cell metaplasia should be considered as an innocuous alteration of the endocervix, probably representing a terminal growth stage of reserve cells.

Atypical Reserve Cell Hyperplasia

This abnormal proliferation of endocervical reserve cells could be defined as a miniature subcolumnar carcinoma in situ. Indeed, when the respective morphologic features of atypical reserve cell hyperplasia and intraepithelial carcinoma are compared, numerous similarities are observed (Figs. 9, 11). The outstanding differences, however, which are more manifest in the tissue section than in the exfoliated cell preparation, are undoubtedly the persistence of an attenuated columnar epithelium overlying the atypical reserve cells and a lesser stratification of the cellular elements in reserve cell hyperplasia. The cellular arrangement, the cytoplasmic and nuclear patterns, and the number of mitoses, are rather strikingly analogous in both lesions.

An incipient and an advanced stage (fringe lesion) of atypical reserve cell hyperplasia are demonstrated in Figs. 8 and 9. In the first illustration a sharp zone of transition is observed between the normal columnar epithelium and the sudden growth of atypical reserve cells. In both illustrations the newly formed abnormal epithelium is composed of four to six layers of rather uniform cells lacking differentiation from base to surface. The cellular elements are compact, frequently appear to overlap, possess indefinite margins, and tend to proliferate in a parallel plane. The cytoplasmic mass is homogeneous and scanty. The nuclear chromatin is coarsely distributed and occasional mitoses are noted. A distinct but somewhat flattened columnar epithelium of mucus secreting type can be indentified as precariously attached to the surface.

Although it is unknown what percentage of incipient or marked atypical reserve cell hyperplastic lesions mature to squamous metaplastic epithelium or progress to in situ carcinoma, a cursory statement that most intraepithelium cancers, as seen in Fig. 12, are preceded by such atypical reserve cell proliferation, may not be premature. Wheeler and Hertig strongly suggested this histogenetic pattern[1], and others also have indirectly implicated such a sequence of events.

When marked atypical reserve cell hyperplasia is observed in cone biopsies of the cervix, a complete blocking of the tissue should be done to exclude an associated in situ carcinoma. In any means, the patient should be followed and exfoliative cell studies performed in order to exclude the occurrence of epithelial cancer at a later date.

Carcinoma In Situ

The term "carcinoma in situ" of the endocervix, interchangeably used for "intraepithelial carcinoma," literally refers to an anaplastic intraepithelial process whose component abnormal cellular elements are analogous morphologically to those observed in outspoken invasive cancer, and whose growth is still limited to the superficial endocervical mucosa and/or to that lining the glandular spaces[6].

Our present knowledge of the various morphologic features believed to be characteristic of carcinoma in situ was obtained mostly by the study of intraepithelial lesions known to have preceded or found to be coexistent with invasive cancer. Although not without shortcomings, this approach seemed to be the most appropriate. Furthermore, subsequent experimental observations disclosed that induced preinvasive cancer in the cervix of the mouse possessed similar characteristics.

These inherent features of carcinoma in situ are those of a marked anaplasia of its epithelial components. As shown in Fig. 12 there is undifferentiation from basal to superficial surfaces. The cells are arranged in vertical and compact rows, and as the upper stratum is reached there is an absence of dyskeratosis. The cytoplasmic outlines are obliterated due to excessive cellular proliferation. These abnormal cells detach readily into the overlying open lumen, as seen in the illustration, a phenomenon related to decreased cohesiveness. The nuclei are usually invested with a coarse chromatin network as demonstrated in Fig. 13, and various phases of mitotic activity are encountered, including an occasional atypical so-called "three group metaphase" figure. Unlike the reserve cell hyperplastic lesion, carcinoma in situ is depleted of any overlying attenuated columnar epithelium. A delimiting membrane separates the connective tissue stroma from the tumorous epithelium, which, however, easily detaches when traumatized.

Carcinoma in situ of the endocervix is frequently not manifest on gross visualization, since it often occurs in unicentric or multicentric foci deep in the canal or glands. For this reason, its detection may be unfortunately delayed if the available

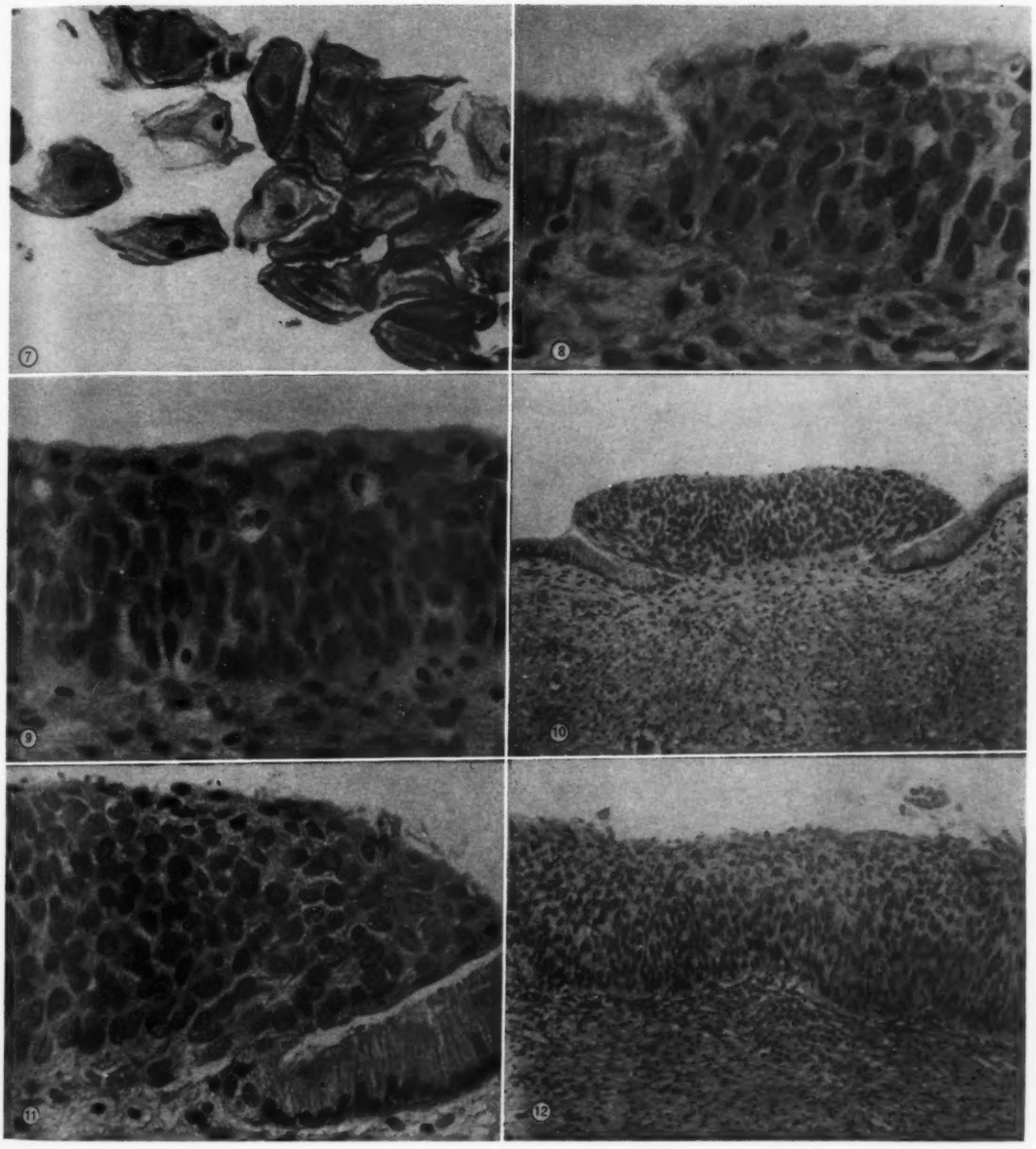


Figure 7. Squamous cell metaplasia. The cells have a pale endoplasm with dense zone as observed in the tissue section.

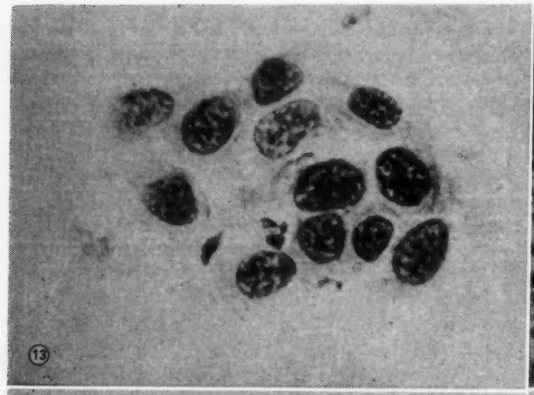
Figure 8. Reserve-cell hyperplasia. Note transition to zone of reserve-cell hyperactivity. Two mitoses are observed among the cells which displace the endocervical columnar epithelium.

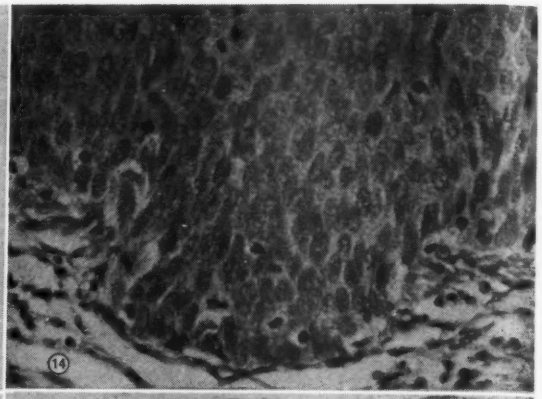
Figure 9. Reserve-cell hyperplasia. Hyperplasia more pronounced than in Fig. 8. Cellular elements closely resemble those of carcinoma in situ.

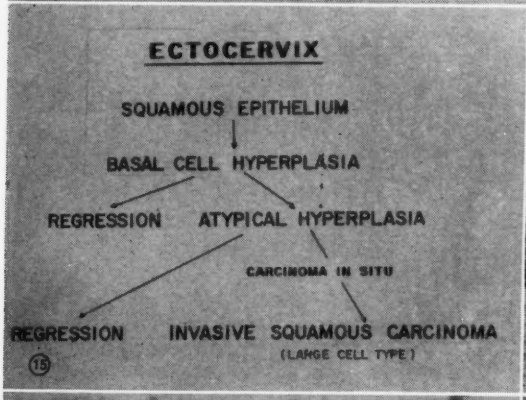
Figure 10. Carcinoma in situ. Well demarcated early endocervical carcinoma in situ with active cellular proliferation.

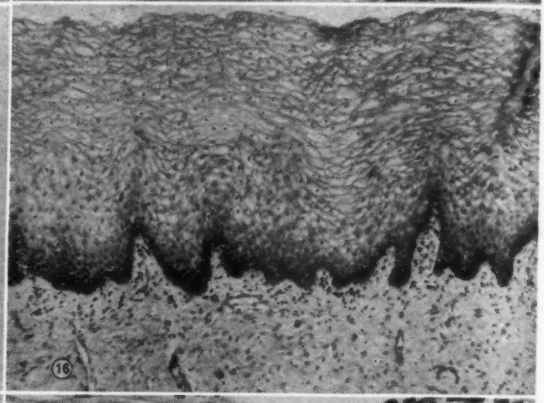
Figure 11. Carcinoma in situ. Abrupt transition. The cellular elements show no evidence of maturation from base to surface. Multicentric foci were observed in this case.

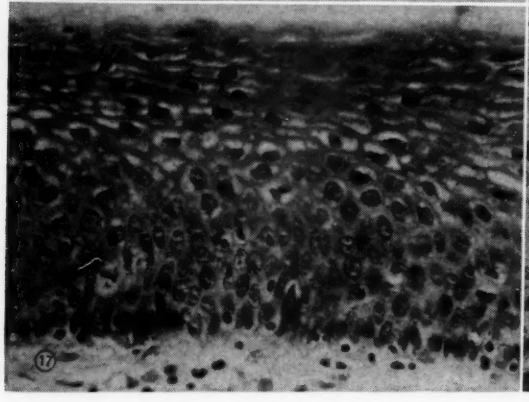
Figure 12. Carcinoma in situ. More diffuse changes with scattered atypical mitoses.











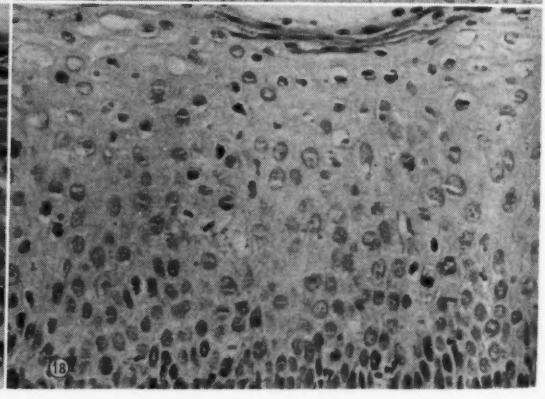


Figure 13. Carcinoma in situ. Exfoliated cells showing an ill-defined cytoplasmic margin with enlarged round to oval nuclei varying slightly in size.

Figure 14. Carcinoma in situ. Note mucosal alterations of carcinoma in situ with prominent mitoses. The basement membrane is distended by ingrowing cells which retain the same axis as the overlying cells. This stromal compression should still be considered as in situ carcinoma, since no definite separate strands are observed as in invasive cancer.

Figure 15. Histogenesis of invasive carcinoma of ectocervix.

Figure 16. Ectocervical epithelium. Normal squamous mucosa showing a deep row of basal cells.

Figure 17. Basal-cell hyperplasia. Multilayered growth of basal cells with prominent chromocenters. Superficial maturation.

Figure 18. Dysplasia (atypical hyperplasia) of squamous epithelium. Although the component cells are immature, there is evidence of maturation towards the surface. Parakeratosis is observed.

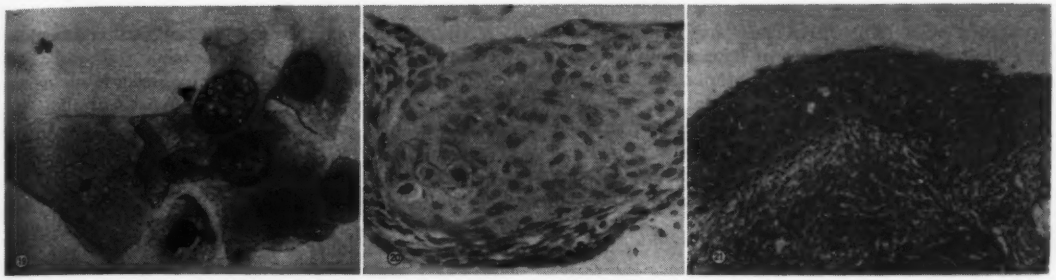


Figure 19. Dysplasia. Cellular elements derived from lesion demonstrated in Fig. 18. The cells are larger than in situ cancer, and the nuclear-cytoplasmic ratio lesser. The cell outline is distinct.

Figure 20. Dysplastic and dyskeratotic changes observed in squamous epithelium of ectocervix overlying a differentiated invasive carcinoma.

Figure 21. Carcinoma in situ of ectocervix. Large cell type of carcinoma in situ of the ectocervical mucosa associated with adjacent invasive carcinoma in other sections. Individual cell keratinization in superficial layer.

routine cytologic screening procedures are not performed. Punch biopsies of the cervix are totally unsatisfactory as a diagnostic method in most instances and wider sampling by sharp cold-knife conization is preferred.

Early Invasive Carcinoma

In the past, clinicians have probably overstressed the importance of the term "early invasive carcinoma" in an attempt to stage more accurately the carcinomas of the cervix into group O or group I lesions, and possibly in order to treat either in a drastically different manner. As a result, cytopurists have been splitting basement membranes by early invading cords of tumor cells in degrees of "questionable early invasive," "probable early invasion," "early invasion not excluded," etc. The resulting confusion more than perturbed some of the one-minded therapeutists.

Early invasive carcinoma of the endocervix should be diagnosed as such only when clusters of cells are identified as separate from the overlying tumorous epithelium and surrounded by a haphazardly arranged connective tissue stroma. The stroma should be penetrated rather than simply compressed by a small intrastromal protrusion from the adjacent mucosal in situ cancer (Fig. 14).

Generally the invading carcinoma retains the cellular characteristics of its overlying mucosal progenitor, be it a large or small cell type of carcinoma.

2. Histogenesis of Invasive Carcinoma of Ectocervix

Invasive carcinoma of the ectocervix arises from the covering squamous epithelium which normally terminates abruptly at the external os. Ectocervical squamous carcinoma is encountered less frequently than that of the endocervix and it is generally more differentiated toward squamous hyper-maturation, comprising various degrees of abnormal keratinization.

This observation leads us to believe that the anaplastic intraepithelial alterations of the squamous epithelium of the portio, which precede invasive squamous carcinoma, may not be as undifferentiated as those of intraepithelial carcinoma in situ of the endocervix. Indeed, in some instances, but not necessarily in all, the mucosa overlying strands of invading differentiated squamous carcinoma shows varying degrees of atypicalities and dyskeratoses (Fig. 20) which per se would not warrant suspicion of impending or existing stromal invasion. However, anaplastic mucosal changes may be more pronounced as in figure 21, but there, also, superficial maturation is evidenced and individual round cell keratinization is noted.

Figure 15 demonstrates a suggested sequence of mucosal modifications which may antecede invasive squamous carcinoma of the ectocervix. The term "basal cell" is purposely used to contrast the basal cellular elements of the squamous epithelium of the ectocervix and the reserve cells of the endocervical mucosa.

Basal Cell Hyperplasia

This growth consists of an abnormal proliferation of the basal cells of the squamous mucosa into varying layers which, however, rather abruptly terminate and abut to maturing squamous cells of the zona superficialis. Although occasional mitoses may be identified, they are infrequent and the basal cells tend to be uniform, ill-delineated, and their nuclei enlarged. The chromatin is coarse and nucleoli are noted in areas (Fig. 17). This growth process probably regresses to normalcy in most instances, but occasionally may progress to dysplastic changes referred to as atypical hyperplasia of squamous epithelium.

Atypical Hyperplasia of Squamous Epithelium

Such a lesion seems to represent an excessive basal cell activity by which the entire thickness of the epithelium is replaced by multilayered basal cellular elements showing, however, more or less differentiation as the surface is reached (Fig. 18). There is generally a superficial parakeratotic layer with focal hyperkeratosis in some instances. The altered cells obtained by scraping the ectocervical

mucosa can easily be differentiated from normal squamous cells (Fig. 19). These atypical cells retain the polygonal contours of squamous cells, but are usually smaller, and the cytoplasmic margins are well-defined. Although the nuclei are very prominent and large, the nuclear-cytoplasmic area ratio is much less than that observed in cancer cells. Dyskeratotic elements are present in the spreads as well, although not demonstrated in the illustration.

Atypical hyperplasia of squamous epithelium is known to persist for years and even recur intermittently. It has only seldom been reported to finally develop into carcinoma. Women who harbor such a lesion should be followed annually in the sense that stimuli, which provoke these ectocervical changes, may be responsible for more adverse anaplastic alterations within the endocervix.

Carcinoma In Situ of Squamous Epithelium

Intraepithelial carcinoma of the ectocervical squamous mucosa comprises cellular components similar to those of the endocervical in situ cancer. However, the degree of undifferentiation tends to be lesser; the anaplastic cells are usually larger and prosoplastic changes are noted. The terminology of keratinizing carcinoma in situ would appear to be justifiable for most cases of ectocervical intraepithelial lesions.

Summary

An attempt has been made to present the various epithelial alterations of the endocervical and ectocervical mucosae which may be precursors of invasive carcinoma of the cerix uteri. Although the changes less than marked epithelial anaplasia knowingly revert to metaplasia or normalcy, some obviously are progenitors of in situ carcinoma itself.

An appraisal of the clinical importance of hyperplasias, dysplasias and anaplasias followed their morphological description. There still exists controversy among pathologists as to what constitutes a true carcinoma in situ and numerous erroneous statistical figures as to its prognosis are continuously submitted in various publications.

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Surgery

The Post-Surgical Room

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This number of the Review, being the effort of the Medical Staff of St. Boniface Hospital, would be remiss indeed not to mention a facility that is unique to this institution — the post-surgery room. It is the only one in operation in Winnipeg and one of very few in all of Canada.

In the past two decades successful advances in surgical technique have occurred as a result of progress in a number of fields. First and foremost in permitting extirpation of more and more of one's anatomy has been the development of safe anaesthetic agents and of the training of experienced anaesthesiologists to administer these properly. To a lesser degree, but nonetheless important has been the proper understanding of the basic physiology of the "internal milieu"; of the replacement of lost fluids and electrolytes; and of adequate shock therapy with readily available large quantities of whole blood. In addition, the discovery of a host of antibiotics has prevented deaths from secondary infection in otherwise successfully surgically treated patients.

The management of the surgical patient does not end with the insertion of the last skin suture. Nor are patients stretched out on a cot and wheeled unconscious to their bed and left in the care of inexperienced personnel while they awaken. Modern usage demands their proper transportation to a recovery room where they are cared for until they are awake and coherent before being sent to their ward.

Recovery rooms fall into two categories. One is the short-stay or post-anaesthetic room and the other is the long-stay or post-surgical room.

The post-anaesthetic room, as its name implies, keeps patients until they have recovered from the effects of the anaesthetic, are fully conscious and are cognizant of their whereabouts. This unit is under the supervision of the Department of Anaesthesia. It is well staffed with experienced nurses, aides and orderlies and is properly equipped with oxygen, suction, emergency drugs, etc. It naturally is part of the operating theatre. This unit is only open for about an eight hour period and has its peak load about noon.

The second type of facility, the post-surgical room or the intensive medical treatment unit as it is being called in some places (especially when non-operative cases such as diabetic coma patients, accident cases, severe stroke cases are being treated) is one that is not too commonly seem as yet. At

St. Boniface Hospital we are fortunate in having both types of facility. In this hospital this room is reserved solely for surgical cases.

The unit consists of 16 beds and is staffed by a total of 16 graduate and student nurses and orderlies. Each bed is fully equipped with suction, oxygen, etc. (see photograph). The layout is such that the supervisor is in constant contact with all her patients and can command instantaneous attention.

The average stay of patients in the post-surgical room is longer than the two hours or so that are spent in the post-anaesthetic room. (After hours when the short-stay room is not staffed, this room acts as a short-stay room for emergency operative cases. There were 250 such admissions during 1958, with an average stay of 1.15 hours).

Usually, all major surgery is sent to this room with the majority of the occupants being thoracic, extensive abdominal or neurosurgical cases. Dependent on the types of cases, and the complications that develop, the average stay varies tremendously (see Table 1). This table lists the ten longest stay type of cases in decreasing frequency. The longest stay patients were chest, followed by bowel obstruction and resection; then comes stomach and duodenum, neurosurgery and colon and rectum. Not shown in these average studies are the few cases that may have stayed for periods up to 600 hours.

TABLE I
Analysis of Ten Commonest Admissions to Post-Surgical Room in 1958

Type of Operation	Number of Cases	Average Stay (Hours)
Chest (heart and lung)	67	113.6
Bowel resection and obstruction	36	113
Stomach and duodenum	142	94
Vascular surgery	13	93.5
Neuro-surgery	41	91.6
Colon and rectum	83	77.9
Kidney and ureter	30	45.8
Gallbladder and bile ducts	237	44.6
Hysterectomy	89	34.9
Bladder and prostate	126	31.7

It is obvious that the grouping together of post-operatives must necessarily result in better care of these patients and we have outlined what we feel to be the most important benefits and detractions of this facility.

Advantages

1. Proper supervision by trained personnel.

The decrease generally in numbers of nurses has naturally resulted in a decrease in private duty nurses. This has increased the work of ward supervisors with a lessening of availability and experience of duty nurses. More extensive specialized techniques require special post-operative care that many private duty nurses are unaware of. In this unit the patients get excellent 24-hour care by personnel who are trained in the modern modalities of care.

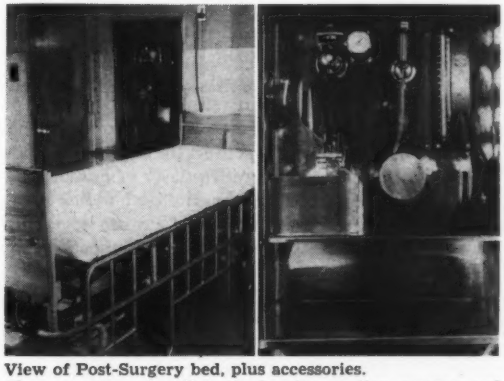
2. Emergency equipment is available at every bed.

Oxygen, suction, etc. are present. The nurses carry out proper breathing exercises, movement and exercise and this results in a decrease of pulmonary and other complications.

3. Complicated suction apparatuses are used in major thoracic, intestinal and urologic surgery. Many nurses do not understand their use, but in this unit the personnel are quite familiar with all the various suctions and drainages.

4. The staff have only a few patients to look after and can do so most advantageously as they never have more than 100 feet to walk for supplies and materials.

This all results in the patients' post-operative recovery being hastened and their convalescence shortened.



View of Post-Surgery bed, plus accessories.
Close-up view of wall unit, showing suction oxygen and sphygmomanometer.

Disadvantages

From a patient's standpoint the disadvantages are:

1. The presence of a number of other equally sick people in close proximity.

2. Restricted visiting hours by family and friends.

3. Many private ward patients dislike the routine and resent taking orders, having in the past being used to ordering a special nurse about.

4. Patients become depressed if their stay is too long because they feel that they must be extremely ill to warrant this special care.

It can be seen that the advantages far outweigh the disadvantages and of necessity benefit the person whom we are all striving to help—the patient.

No doctor who has had occasion to avail himself of the services of the unit has ever regretted its installation and most important, as has been said, the development of the recovery room has been likened by some to the greatest single advance in the safeguarding of the surgical patient since the discovery of anaesthesia.

Changing Concepts in the Extent of Resection for Carcinoma of Lung

James S. McGoey, M.D.
Department of Surgery, St. Boniface Hospital

The increased tendency to utilize lobectomy as the procedure of choice in the attempted surgical cure of some bronchogenic carcinomas, is receiving increasing recognition. A quotation from the opening paragraph of a paper presented by Robinson, Jones and Meyer[1] at the American Association for Thoracic Surgery in May 1956, is significant:

"In presenting this subject for discussion by the members of the Association, we should like to have it clearly understood that we are not raising the question of whether lobectomy is a better operation than pneumonectomy in the treatment of pulmonary carcinoma, but rather, in treating a human being for carcinoma of the lung, under what circumstances is it preferable to remove less than the entire lung? We believe that every surgeon worthy of the name must, in treating a given patient, temper the zeal of the surgical technician with the judgment of the physician within him. His aim in treating a patient in such a manner as to make that life as happy and satisfactory in the living as is possible. Because of the multiplicity of clinical factors which bear upon each individual patient, "indications" for surgical procedures must serve not as absolute criteria but rather as guideposts to aid the surgeon in reaching a decision."

The question as to whether to perform lobectomy or pneumonectomy, or even radical pneumonectomy is not easily answered. In the past, and especially among the theorists and uninitiated, emphatic statements were made that pneumonectomy was the operation of choice, and that lobectomy was justifiable only in patients who would not survive a more complete operation. However, in more recent years, thoracic surgeons are impressed by the evidence which has accumulated to show that lobectomy is the operation of choice under a wider range of circumstances.

Up until about 1950, the question of lobectomy for carcinoma of the lung had not caused much controversy. About that time, it became apparent that there was a change evolving in the surgical attitude as to the extent of resection for bronchogenic carcinoma. Churchill[2] in 1950, was among the first to publish a report establishing that lobectomy had some merit in the curative treatment of carcinoma of the lung. This was somewhat contrary to the general trend in the surgical treatment of malignancies elsewhere in the body, where resections were becoming progressively more radical and extensive; and have remained so. Thus the history of the extent of the surgical treatment of bronchogenic cancer has differed from that of other malignancies, since the first successful resection in 1935.

Bronchogenic carcinoma has continued to have an unfavorable prognosis, when all cases are considered, resectable and non-resectable. There are two general ways in which results can be improved, (1) more radical resection, and (2) earlier diagnosis.

More radical "en bloc" resections have given improved results in other malignancies. However, this has not been the case with bronchogenic carcinoma. Those who advocate "radical pneumonectomy," are trying to apply to the lung the same techniques as are applied in carcinoma of the left colon, or carcinoma of the breast, with resection of the lesion and en bloc resection of the lymphatics. However, the lung differs markedly from these other organs, as regards both variable lymphatic drainage of the lobes and also the site of the lymph nodes in the mediastinum. For example the normal drainage of the left lower lobe is to the lymph nodes of the right upper mediastinum. Also because of the widespread intercommunication of the lymph channels from the two lungs, it is questionable whether a widespread removal of the nodes, accomplishes what is theoretically desirable. Added to these disturbing facts, is the definite increased morbidity and mortality experienced with these more radical procedures . The so-called "radical pneumonectomy" as described by Cahan, Waters and Pool in 1951 has been performed by many thoracic surgeons. Enthusiasm since that time, especially among those who have performed these operations has waned. No series of cases has been forthcoming with long term survivors which substantiates that extended operation has anything to offer over the conventional intrapleural operation.

Numerous groups have now reported on this increased tendency to use lobectomy rather than pneumonectomy for the procedure of choice. Overholt has reported that prior to 1950, lobectomy was carried out in only six per cent of resections, while since that time 35 per cent of resections are now lobectomy. Robinson, Jones and Myer[1] report that the percentage of resections done by lobectomy rose from 30.4 per cent prior to 1950, to 61.5 per cent in 1955. These authors also pointed out that the three-year survival rate was 71.4 per cent in patients treated by lobectomy and 16.3 per cent in those patients treated by pneumonectomy. Another important factor which is paralleled in most institutions is brought out by Kirschner's reporting on the results in the Bronx Veteran Hospital, showing that in a total of 113 resections, there has been no mortality in the lobectomy group, as compared to 16.9 per cent in the pneumonectomy group. For the past four years we have found ourselves utilizing lobectomy more frequently than previously. Of course, lobectomy has never been insisted upon in a situation where pneumonectomy is indicated because of the site or the extent of tumor. It would

be to defy the basic tenets of our surgical pathology to attempt to utilize lobectomy where it is obviously not the operation of choice.

There is yet another factor which is leading us towards the greater utilization of lobectomy, and that is the willingness by the surgeon now to accept older patients for surgical treatment. Many of these patients could not even be considered for total pneumonectomy, but will withstand lobectomy quite well. Not only are surgical techniques improved from former years, but equally important are the better anaesthesia, the new techniques and apparatus for pre and post-operative pulmonary care, the post-operative surgical departments, the availability of antibiotics, and adequate blood transfusions.

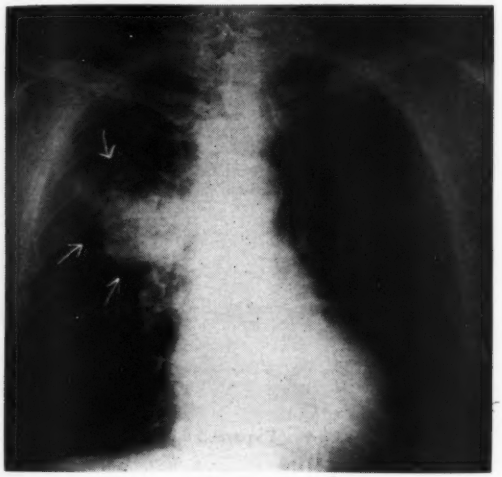


Figure 1

Case Reports

Case 1. — T. J., a 61 year old man was admitted to St. Boniface Hospital on December 3, 1955 for an investigation of opacity in the right lung which had been revealed on routine chest x-ray. Patient had no symptoms referable to his respiratory tract. He had had a heart attack in 1946. The rest of the history is of no significance. On investigation his chest moved equally and symmetrically on both sides. On auscultation the breath sounds were found to be broncho-vesicular in type over the left upper lobe, and there was some question of breath sounds being decreased in intensity over the right upper lobe. Heart sounds were normal. Blood pressure 132/90. On December 3 the patient was bronchoscoped. Carina and left bronchus were noted to be normal. Mucosa bled readily around the right upper lobe orifice. A biopsy was taken which revealed subacute bronchitis and washings taken at the time of bronchoscopy revealed malignant tumor cells. Provisional diagnosis was either adenocarcinoma or bronchogenic carcinoma. Chest x-ray (Fig. 1) on December 4 revealed a diffuse irregular increased density at this superior portion of the right hilum which extends into the anterior segment of the right upper lobe on lateral view. The appearance is most suggestive of a bronchogenic carcinoma. An electrocardiogram on December 7 suggested pulmonary hypertension. This was repeated on December 12, at which time an incomplete bundle branch block was reported with further suggestion of pulmonary hypertension. On December 9 patient was taken to the operating room for thoracotomy which was not performed at this time because of a persistent low blood pressure after the induction and being turned to his left side. Patient was next taken to the operating room on December 14 at which time right thoracotomy was performed. There was a firm mass which appeared to be carcinoma which involved the right upper lobe. It did not extend to the middle lobe, and there was no extension into the main branches. Decision was made that a right upper lobectomy would be just as curative in this case as a pneumonectomy. Pathological report on the surgical specimen was anaplastic epidermoid carcinoma of the lung, grade II. Patient's condition was considered satisfactory and he was discharged from hospital on December 27, 1955.

This patient is in good health and continuing with his work at last report in March 1959, with no evidence of spread of disease.

Case 2. — C. M., a 55 year old man was admitted to St. Boniface Hospital on December 7, 1955. His chief complaint on admission was that he had been spitting up blood for the last ten days. He stated that he had previously coughed up blood in May, 1955 during an episode of pneumonia. Blood at this time was dark in color and lasted about two weeks. Following this, he was completely well until November 26, 1955, when he noticed a faint bloody tinge on his sputum after coughing. In the morning he generally coughed up one teaspoonful of blood. He is a heavy cigarette smoker. In 1949 he had a gastrectomy. The diagnosis at the time was carcinoma of the pyloric region of the stomach. He had had an apparent cure of the carcinoma of the stomach. On examination he was noted to be in no respiratory distress, but occasionally coughing during the examination and his sputum was tinged with blood. Left lung had crepitation at the left base. The rest of the physical examination was within normal limits. Bronchoscopy was performed on December 7. It was noted that the mucosa bled readily in the left lower lobe and aspirations were taken from this region. Smear for tumor cells revealed the presence of abnormal cells which were suggestive of malignancy. Chest x-ray on December 7 (Fig. 2) showed a faint haziness extending out from the left hilum into the left lung field and another small area of haziness immediately below and lateral to the left hilum,

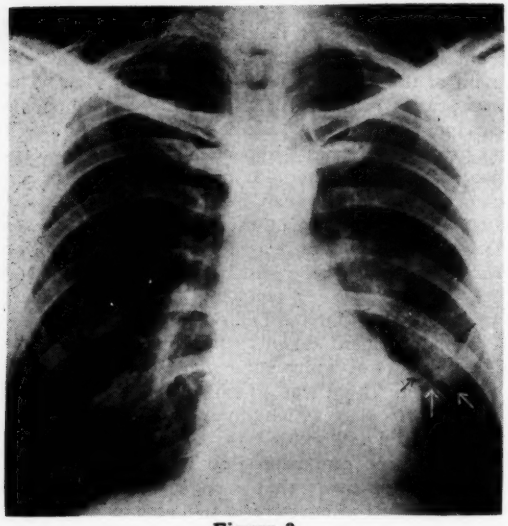


Figure 2

about 1½ inches in diameter. At operation on December 10, 1955, the lung was examined and decision made to do a left lower lobectomy. Pathological report was anaplastic bronchogenic carcinoma. Post-operative course was uneventful, and he was discharged from hospital on December 21, 1955. This patient has been seen at frequent intervals. When last seen in January 1959, patient was apparently well and free of disease.

This is the case of an interesting patient who is already a five-year survival from carcinoma of the stomach and is now more than a three-year survival from carcinoma of the lung. He is able to carry on with his vigorous outdoor work with the Telephone Company.

Case 3 — A. C., a 65 year old man was admitted to St. Boniface Hospital on December 10, 1955, complaining of cough and shortness of breath for six months. Patient had a long history pertaining to his respiratory tract, beginning with exposure to gas during World War I. He has been employed as a fireman for the last 35 years. His first symptomatic trouble began in 1947, when he developed severe bronchitis. Since that time he had considerable cough during the winter months with some sputum. At the same time his shortness of breath has been becoming progressively worse. Several weeks prior to admission he developed a fever to 103 for several days. This was treated with some relief. Symptoms of shortness of breath persisted and he was admitted to hospital for further investigation and treatment. Past illnesses include a cholecystectomy in 1953. The rest of the history is not significant. On examination the patient is obviously short of breath. Chest is symmetrical in shape, but the movements and breath sounds on the left side are diminished. Blood pressure is 115/60. There were no murmurs. The heart is enlarged and the rhythm is irregular. The rest of the physical examination is within normal limits. Patient was treated initially as cardiac failure and bronchitis. However in consultation it was suggested that his fever and rapid sedimentation rate were not explained on the bases of cardiac disease and that possibility of carcinoma of the lung should be considered. Consequently he was bronchoscoped January 4, 1956. No tumor was seen, but a rough membrane in the left lower lobe bronchus bled easily. An abscess below this was drained. Pathological report of the cell block from the bronchial aspirations was compatible with bronchogenic carcinoma. X-ray examination (Fig. 3) of the chest

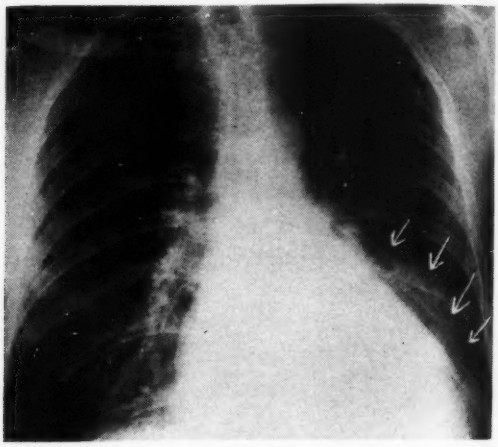


Figure 3

suggests a density in the region of the left hilum which could be compatible with bronchogenic carcinoma. Electrocardiogram on January 16, 1956, shows auricular fibrillation with evidence of ischemia and hypertrophy of the left ventricle. Patient was taken to the operating room on January 20, 1956, for thoracotomy. Pre-operative diagnosis was carcinoma of the left lower lobe. At operation the left upper lobe was found to be well expanded. The left lower lobe was completely atelectatic. There were some large lymph nodes in the region of the left lower lobe extending into the mediastinum. It was felt that resection of the lung should be done. However on clamping the bronchus patient did not seem to respond well and decision was then made to do a left lower lobectomy. Pathological report —infiltrating epidermoid carcinoma of the lung, grade III involving regional lymph nodes. His post-operative course was relatively uneventful. He was discharged from hospital on February 9, 1956. This patient had some subsequent urinary difficulties. He remained reasonably well until he was admitted to another hospital with evidence of cerebral metastases, and he died on July 17, 1957.

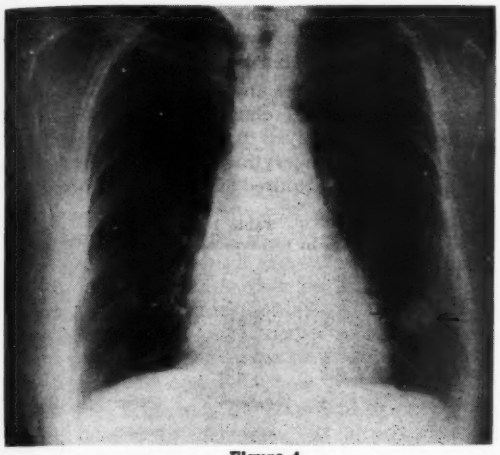


Figure 4
This is the x-ray of an asymptomatic 69 year old female, with a small "coin" lesion, discovered on routine film. Left lower lobectomy was carried out for malignant lesion with complete recovery, in 1956.

The patient falls into the older age group in poor general condition. He suffered from cardiac enlargement with auricular fibrillation. He lived for 18 months following resection of a completely atelectatic left lower lobe with abscess, after it was demonstrated in the operating room that this patient would not tolerate a pneumonectomy.

Summary and Conclusions

We feel now that lobectomy, properly applied, is, because of what has been outlined above, as radical as pneumonectomy in bronchogenic carcinoma; and this fact combined with its inherent low operative mortality rate, low morbidity rate, and better functional results, recommends it for continued use in the surgery for this disease. At the present time, the following facts are suggested.

Lobectomy should be done when (1) the patient is a poor surgical risk, due to low pulmonary reserve, or general disability, (2) the patient has a small, isolated and peripherally located tumor nodule, (3) the patient has a well defined, localized bronchogenic carcinoma, with hilar and local nodes not involved, and (4) the patient cannot be cured, but lobectomy would afford good palliation.

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Biochemistry

The Detection of Intravascular Hemolysis

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Department of Biochemistry, St. Boniface Hospital and Department of Physiology, University of Manitoba

It is the purpose of this paper to draw attention to two relatively new methods for detecting intravascular hemolysis and for assessing its severity. The first of these, which we call the "plasma hemochromogen," is a measure of the concentration of hemoglobin and its immediate breakdown products in plasma. The second test is based on paper electrophoresis of serum to detect the presence of free hemoglobin, its degradation product, methemalbumin, and of the alpha$_2$-globin which binds hemoglobin liberated by hemolysis.

It should be emphasized that these procedures merely supplement and, certainly, cannot replace the conventional methods of diagnosing hemolytic disease described in hematology texts[1] and monographs[2]. However, there are cases of undoubted intravascular hemolysis in which the serum bilirubin and the urinary urobilinogen findings are equivocal, the Coomb's test negative and the reticulocyte increases minimal. Quantitative fecal stercobilin and Cr^{51} labelled red cell studies can be of help in such patients, but these procedures are relatively cumbersome and are not readily applicable when hemolysis is transient. In such cases the two new tests can often be diagnostic.

Methods

The plasma hemochromogen is determined according to Hunter et al[3]. The technique is rapid and simple, but great care must be taken to avoid mechanical hemolysis during blood collection.

The electrophoretic method we use to identify hemoglobin, the hemoglobin-binding-alpha$_2$-globulin and methemalbumin in serum is essentially that of Nyman and Laurell[4, 5], except that we have simplified the staining by using "Hematest" tablets dissolved in water as suggested by Dr. Paul T. Green.

Applications

The plasma hemochromogen level normally is less than 15 mgm.%. It rises immediately after intravascular hemolysis to a level that reflects the severity of the hemolytic process and may reach 500 mgm.% or more. Suppression of the hemolysis is followed by a rapid fall in the plasma hemochromogen, although normal levels are not reached for 5 to 7 days. Because of this delay, it is often possible to detect a severe bout of hemolysis some days after the acute episode. Also, with this technique, alert technicians have occasionally been able to detect unsuspected hemolysis in patients whose serum, though yellowish-green in color to the eye, has a relatively low bilirubin level.

The serum electrophoresis method of identifying heme pigments is somewhat more sensitive than the hemochromogen procedure. The technique and, particularly, the interpretation of the electrophoretic strips are, however, more complex. The alpha$_2$-globulin of normal serum is able to bind between 30 and 130 mgm.% hemoglobin. This complex with hemoglobin can be readily identified and

is a very sensitive indicator of minimal hemolysis. This hemoglobin-alpha$_2$-globulin complex is removed from the circulation within two days. The alpha$_2$-globulin lost in this manner is slowly replaced in the plasma over a 5-7 day period following the hemolytic episode. This temporary deficiency of the alpha$_2$-globulin can also be detected electrophoretically by the decreased ability of the subject's serum to bind hemoglobin added to it. Thus, like the plasma hemochromogen test, the electrophoretic method is able to detect relatively mild hemolysis some days after it has occurred.

When hemolysis is severe and/or persistent, the same sequence of appearance and then disappearance of the hemoglobin-alpha$_2$-globulin complex from the serum occurs, but is followed by the appearance of "free" hemoglobin and of another heme pigment, methemalbumin, on the electrophoretic strip. It is the methemalbumin that gives the plasma in severe hemolytic disease its characteristic brownish-green colour that is quite different from that of bilirubin. All of these heme pigments, of course, react in the plasma hemochromogen test. The latter, because of its simplicity, is therefore preferable for following the course of an established case of hemolysis. However, as the hemolysis subsides, only the electrophoretic method can detect the persistence of minor degrees of red cell destruction by a failure of the hemoglobin-binding alpha$_2$-globulin to return to normal levels in 5 - 7 days. The only condition known to interfere with the electrophoretic test at this stage of the hemolytic process is liver disease which causes a chronic low level of the hemoglobin-binding globulin.

Table 1 summarizes the findings of the plasma hemochromogen and electrophoresis tests in the intravascular hemolysis that occurs in association with neoplasms, in erythroblastosis fetalis and in various types of acquired hemolytic disease.

Table I
Findings in Intravascular Hemolysis

Intravascular hemolysis	Plasma hemochromogen (mgm.%)	Alpha$_2$-globulin that binds hemoglobin	Hemoglobin-Alpha$_2$-globulin complex	Methemalbumin
None	5 - 15	present	absent	absent
Mild	15 - 30	subnormal	present	slightly positive
Moderate, persistent	30 - 60	absent	absent (usually)	positive
Severe	60 - 500	absent	absent	strongly positive

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Editorial

S. Vaisrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

Guest Editorial

This complete issue of the Manitoba Medical Review is being presented to its readers by the members of the Medical Staff of St. Boniface Hospital. This hospital, the oldest on the Canadian prairies, has witnessed a great many changes in its eighty-nine years of service, but undoubtedly, the greatest change occurred on July 1, 1958, when the new Manitoba Hospital scheme came into being. Many problems were solved by this universal, compulsory scheme and some new ones were created.

For over a century the Sisters of Charity (Grey Nuns) have devoted themselves to the care of the sick in this Province and in 1871 opened their first hospital.

In the years that followed, the hospital developed from the humble four beds to an imposing 700 beds and in 1922 became officially affiliated with the University of Manitoba as a teaching hospital. The underlying principles of service to the patients, however, have remained unchanged. The administration has always tried to have an institution in which the doctors would find a high quality workshop in which the sick would receive the highest standard of medical care at the most reasonable cost. Because of the dedication of their lives to this work, the Sisters succeeded in realizing these objectives while at the same time promoting the spiritual welfare of the patients, in which they, naturally, were interested.

Well over a million patients received the benefits of the great experience which the Sisters have accumulated, of the dedicated charity for which they were named and of the excellent care which they fostered.

In 1952 the Sisters started the large expansion and renovation program, which they believed necessary in order to continue their work and which was made possible by the very generous gifts obtained from the public, by government grants and by money borrowed through mortgages on their properties in Manitoba.

On completion of the new wing in 1956 a great surge of new activities took place and the hospital became the centre of medical research, teaching and care for a great many doctors, and the hospital of choice for a large number of citizens.

Less than two years later, the Province accepted to become part of the Federal Hospitalization scheme. The changes brought about by this action involved not only the management functions in all aspects, including planning direction and supervision, but affected also the work of the doctors and the preferences, attitudes and welfare of the patients.

The hospitals were guaranteed payment for care rendered to all insured patients. This appeared to be the answer to all the financial problems of hospitals, especially in Manitoba, where costs were to include depreciation as well as operating expenses. Indeed, this guarantee solved most of these problems, and the hospitals are now perhaps in a more secure situation than they have ever been in the past.

It could not be expected that a plan of this magnitude would be initiated without causing a number of difficulties to those most concerned with its operation. Most of the difficulties stem from the increased demand for hospital beds. While urgent cases are admitted without delay, the more numerous elective cases have to wait much longer for admission. It can be confidently expected, however, that in time these problems will be satisfactorily solved.

It is important for everyone to remember that the hospital plan is now, and will remain, a co-operative effort between the Government, the hospitals, the doctors and the patients. With tolerance, sincerity and honesty from all parties one may look forward to a plan which will assure a high standard of care for all the sick persons who need it, which will promise a just and sound future for our institutions and which will provide the doctors with excellent facilities to carry out their professional work.

While each group has a very important part to play in this partnership, the profession as always should educate itself to all the possibilities of the present trend, so that it may accept a position of leadership in guiding the plan to an acceptable solution which can attract the wholehearted support of all its members as well as of the public.

P. L'Heureux, M.D.,
Medical Director,
St. Boniface Hospital.

Abstracts from the Literature

Peptic Ulceration in Childhood. By E. Goulston. M.J. Australia, r: 46: 150, Jan. 1959.

Peptic ulceration in childhood has been classified into four groups according to age— neonatal (up to one year of age), childhood (up to 10 years) and adult type in later childhood. In five years, 31 children with proven peptic ulcers were admitted to three teaching hospitals in Sydney. Nine children had gastric ulcers (five males, four females) and 22 children had duodenal ulcers (14 males, eight females). There were no neonatal or infantile cases. Fifteen children were under 10 years, and sixteen were between 10 and 15 years. Half the patients presented with hematemesis or melena, two after steroid therapy for asthma. Recurrent periumbilical pain was a feature in 12 cases. Appendectomy had been performed in five patients. Surgery was performed in seven of the 31 patients (bleeding leading to surgery in five, chronic pain in one, and perforation in one). Two patients died. All the patients who bled were group O, Rh positive. Radiologically it appears that screening and spot filming of the stomach and duodenum are more difficult in children. The lesions are shallower and smaller, and the duodenal bulb less easy to demonstrate. Owing to less surrounding reaction, the demonstration of a niche is more difficult. Peptic ulceration should be considered in the differential diagnosis of recurrent abdominal pain in children. The complications of this disease are serious in children and may require early surgery and earlier blood replacement than in adults.

A. G. Rogers.

☆

Normal Bowel Sounds. By G. W. Milton. M. J. Australia, 45: 490 (Oct), 1958.

The study of bowel sounds heard with a stethoscope is described in both experimental animals and humans. In humans each quadrant was listened to for one minute, using a diaphragm stethoscope. The frequency of sounds was charted, and the quadrant noted in which the sounds were loudest. The findings suggest that the majority of bowel sounds originate in the small intestine, and that sounds which originate from the pyloric region may occasionally be heard and that such sounds have characteristics which enable them to be recognized. It is rare to have complete abdominal silence for four minutes, but not uncommon to have loud and continuous sounds in healthy individuals. Increased bowel sounds are defined as a count of 20 or more sounds per minute, in each of the four quadrants of the abdomen in a patient who has been without food for 12 to 18 hours. The counting of bowel sounds is irregular when the sounds are frequent, because of the difficulty of distinguishing one sound from another. In practice, therefore counts of less than five per minute, obtained by different observers, are comparable. If the counts are of much greater frequency an accurate comparison can be made only if the counts are all obtained by the same individual.

A. G. Rogers.

☆

Letter to The Editor

April 23, 1959.

Dear Editor:

As is shown below, the annual number of cases of Infectious Hepatitis being reported in Winnipeg indicates that the prevalence of this virus infection is increasing.

Year	Cases
1952	24
1953	48
1954	51
1955	65
1956	83
1957	82
1958	97

In the first three months of 1959, 44 cases have already been reported.

It also appears that this disease, as seen here, is increasing in severity. Recently two children from the same family, who lived outside of Winnipeg, died at the King George Hospital, as a result of Infectious Hepatitis.

When Infectious Hepatitis occurs in a rooming house, and is reported to the City Health Department, the following notice is distributed by the Health Department to all occupants of the rooming house:

> "This is to notify you that a case of Infectious Jaundice has occurred in this household. You are advised to consult your own doctor in order that you may be given an injection which should prevent you from developing jaundice."

It has come to our attention that not all physicians are advising the administration of Gamma Globulin to the other persons who are household contacts to a patient who has Infectious Hepatitis. Gamma Globulin has proven to be quite effective in the prevention of Infectious Hepatitis if given to household contacts in a dosage of .01 cc. per pound of body weight, and for this purpose is available free to physicians.

It is to be hoped that a physician when called to see a patient with Infectious Hepatitis will realize the importance of immunizing the household contacts.

Yours truly,

R. G. Cadham, M.D.,
Medical Health Officer,
Winnipeg, Manitoba.

Social News

Reported by K. Borthwick-Leslie, M.D.

To Dr. Roy Stewart, Illustrious Potentate of Khartum Temple, A.A.O.N.M.S., Oasis of Winnipeg, our sincere "thank you" for a most enjoyable evening at the Shrine Circus. Even Wyatt Earp, in all his splendor, couldn't compete with the dazzling impressive figure of the Illustrious Potentate!

♦

The Manitoba Branch of the Federation of Medical Women of Canada held their annual Reception and Dinner honoring our 1959 graduates, Drs. Margaret Forke, Iona Fern Hammond and Katrina Nagy last Wednesday, at the home of Dr. Emma Adamson, South Drive, Fort Garry.

An excellent attendance, marvelous dinner and thoroughly enjoyable evening.

The slate of officers for the coming year is: President, Dr. Emma Adamson; Vice President, Dr. Donna Randle; Secretary, Dr. Katrina Nagy; Treasurer, Dr. Aldis Wengel.

The retiring President, Dr. Marie Storrie was honored for her most efficient management of officers in 1959. Great plans are in order for 1960, convention year.

♦

Dr. Glen Hamilton (Major) has been awarded the Canadian Efficiency Medal as announced by Headquarters, Prairie Command in recognition of his military service from C.O.T.C. 1927 through to 1945.

An interesting sidelight is that James Hamilton, scout for the Queen's Own Rifles in the Riel uprising, 1885, was Glen's grandfather, and subsequently overseas. Dr. Hamilton for a time was medical officer to the Queen's Own Rifles.

Dr. Glen is and has been, very active with the Defence Medical Society in Winnipeg.

♦

Drs. F. D. Bertalanffy and K. L. Moore, Dept. of Anatomy, have been promoted from assistant professors to associate professors, Faculty of Medicine, U. of M.

♦

Dr. Marie Cameron, director of the Bible Hospital of the Latin America Mission in San Jose, Costa Rica, has been back in Winnipeg, though I've not seen her as yet. She had been in Jerusalem attending prophetic and medical conferences.

♦

London, England — Large reunion of Winnipeggers, via the Chamber of Commerce Goodwill Tour of the continent. Among those having fun were Dr. Tom Casey (happy, interested and well in his post graduate work there) and Dr. Leonard Bradley, Superintendent of the General Hospital, holidaying in Britain.

Dr. Ida Armstrong has arrived in London by air — northern route — where she will attend the marriage of her niece on June 6th. She plans on joining the rest of members in Edinburgh in July for the convention and remainder of the planned tour. Lucky gal.

♦

James P. MacLaughlin, M.D., M.Rad., D.M.R.D. announces the opening of his office for Diagnostic Radiology at 611 Boyd Bldg., Winnipeg. Telephone WH 2-1836.

♦

Mr. and Mrs. Robert Hesp announce the engagement of their daughter Vivian Tannis to Dr. Martin James Hollenberg, son of Mrs. Abraham Hollenberg and the late Dr. Abraham Hollenberg. The marriage will take place June 18, 1959 in Shaarey Zedek Synagogue.

♦

Mrs. M. W. Colcleugh, Vancouver, announces the engagement of Sandra Jean Wise, daughter of Mrs. Colcleugh and the late Lt. R. A. Wise to David Alexander Goodwin, son of Dr. and Mrs. A. M. Goodwin. The wedding will take place May 30, 1959.

♦

Stork Department:

Dr. and Mrs. D. R. Magee announce the birth of Janice Elizabeth on May 18, 1959.

♦

Dr. and Mrs. Peter Suderman, Estevan, Sask., welcomed Peter Douglas, also on May 18, 1959.

♦

Dr. and Mrs. Jack Rubin announce the arrival of Ronald Hart on May 12, 1959, baby brother for Barbara and Jacqueline.

♦

Dr. and Mrs. R. O. Hinch, Minnedosa, Man., announce the birth of Audrey Diane on May 21, 1959.

♦

Dr. and Mrs. David Arnott, Guelph, Ont., announce the arrival of John David, May 14, 1959, brother for Janice.

♦

Dr. and Mrs. Robert La Freniere, St. Anne, Man., welcome a son, Andre, May 21, 1959, brother for Mona, Robert and Roger.

♦

Dr. and Mrs. F. P. Waugh are happy to announce the arrival of Robin Jack, May 25, 1959.

♦

Dr. and Mrs. James Mitchell are pleased to announce the arrival of Carla Rosslyn, on April 27, 1959, sister for Susan, Sheila and Barbara.

Association Page

Reported by M. T. Macfarland, M.D.

World Health Organization

Founded April 7, 1948

The World Health Organization, one of the largest of the United Nations specialized agencies, with 84 members and four associate members, works for "the attainment by all peoples of the highest possible level of health." It carries out a wide range of programs designed to help countries strengthen their public health services.

Nearly everywhere in the world one of the main obstacles to health improvement is the grave shortage of health workers of all kinds. WHO devotes particular attention to this problem, sponsoring international training courses and seminars, arranging for groups of experts to give practical on-the-spot demonstrations, and granting hundreds of fellowships each year which enable doctors, nurses, etc., to study or undertake research abroad. Subjects of special concern in all these varied activities, which are grouped together as WHO's advisory services, include malaria, tuberculosis, venereal diseases, maternal and child health, nutrition, environmental sanitation, and mental health.

WHO also maintains central technical services, comprising work in such fields as biological standardization and unification of pharmacopoeias, collection and dissemination of epidemiological intelligence, special international research projects on a number of parasitic and virus diseases, and a series of some fifteen types of technical publications. It is also concerned with the health aspects of the use of atomic energy.

WHO's machinery comprises:

the World Health Assembly, the policy-making body of the Organization, with representatives of all member states;

the Executive Board, consisting of 18 technically qualified persons designated by 18 member states elected by the Assembly;

the Secretariat, consisting of the Director-general (Dr. M. G. Candau) and such technical and administrative staff as required.

To keep the Organization up to date in the technical details of its programs and to recommend action on the basis of the latest research discoveries, expert panels of carefully chosen specialists from all parts of the world cover virtually every aspect of health work.

Since WHO's task is to assist, advise, and coordinate—not to function as a supra-national health administration — it has applied the principle of regionalization. Six regional offices are in operation, serving South-east Asia, the Eastern Mediterranean, the Americas, the Western Pacific, Africa, and Europe.

Through these offices the bulk of WHO's operations in the field are carried out. The member countries of each region meet regularly in committee to plan local programs and to review the work of the regional office. These plans then go to Geneva to be fitted into the framework of the Organization's total program.

Close collaboration and liaison on a wide range of matters affecting health standards are maintained with the various organs of the United Nations, other specialized agencies, the United Nations Children's Fund, and a considerable number of international non-government organizations.

Southern District Medical Society

A meeting of the Southern District Medical Society was held at the Altona Hospital on Thursday, April 23rd, 1959.

Present were: Drs. S. S. Toni, Altona, President; and J. P. Boreskie, Gretna, Secretary; E. A. Schabauer, Altona; H. W. C. North, Carman; J. M. Stiglmayr, Emerson; B. J. Froese and H. U. Penner, Winkler; Edward Johnson, Selkirk; J. C. Menzies, Morden, S. Israels, J. P. Maclean and M. T. Macfarland, Winnipeg.

The Scientific Session consisted of papers by Dr. S. Israels on the subject of "Nephrosis" and Dr. J. P. Maclean on the subject of "Endocrinology in General Practice."

At a business session Drs. S. S. Toni, Altona and J. P. Boreskie, Gretna were reappointed President and Secretary respectively, for the year 1959-60.

Dr. Edward Johnson, Selkirk, President of the Manitoba Medical Association brought greetings and discussed such items as Indigent Care Survey, Fee Schedule study by Professional Policy Committee, and hospitalization under the new Manitoba Hospital Services Plan.

Dr. M. T. Macfarland, Executive Director, dealt with legislation introduced at last session of the legislature, and claims being put forth by the aspirants for office in the May 14th provincial election.

Drs. J. C. Menzies and S. S. Toni opened the discussion on the establishment of a Benevolent Fund and study of the Columbus Plan for elimination of Dichotomy.

Following a reception a delicious dinner was served in the dining room, and members dispersed after a pleasant evening.

It is anticipated that another meeting will be held in the fall—prior to the snowstorm similar to that which prevented the holding of the November, 1958 meeting.

M. T. M.

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(1) *ferric iron content is not more than 0.8.mg. and does not increase on prolonged storage.*
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(3) *intolerance is less than 1%.*
(4) *haemoglobin response is 1%—2% per day.*

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CEREVON 'B'

Each capsule/tablet/teaspoonful contains:

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Thiamine Hydrochloride..... 1 mg.
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bottles of 100 and 1,000.

CEREVON 'B' TABLETS
bottles of 100 and 1,000

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CEREVON 'B' ELIXIR
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Winnipeg Medical Society Committee Reports 1958 - 1959

Committee Reports 1958 - 1959

Report of the Secretary

To the President and Members of
The Winnipeg Medical Society:

The first meeting of the Council was held on the 23rd of June, 1958, and regular monthly meetings were commenced on the 22nd of September, 1958, and were held monthly thereafter. Dr. Downey was in the chair for all meetings. During the year nine general meetings were held. Attendance at the meetings continues to be rather disappointing, averaging about seventy members per session. As this is only about ten per cent of the members resident in Winnipeg, it is apparent that there is room for improvement in this regard.

During the past year this Society lost through death the following members:

Dr. G. W. McIntosh
Dr. E. T. Etsell
Dr. M. Hjaltsson
Dr. W. E. Chasney
Dr. W. E. Campbell

An amendment to the constitution has been proposed and will be brought before the meeting.

I wish to express my thanks and the appreciation of the Society to Dr. M. T. Macfarland and his office staff for their invaluable assistance during the past year.

Respectfully submitted,

D. M. Bruser,
Secretary.

Treasurer

Auditors' Report

5th May, 1959.

To the Members,
The Winnipeg Medical Society,
Winnipeg, Manitoba.

In accordance with your instructions we have examined the accounts of

THE WINNIPEG MEDICAL SOCIETY
— and —
THE WINNIPEG MEDICAL SOCIETY LIBRARY FUND

for the fiscal year ended 30th April, 1959, and have prepared therefrom and submit herewith the following financial statements for your consideration:

Exhibit A, Balance Sheet, as at 30th April, 1959,
Exhibit B, Statement of Revenue and Expenditure.

We report thereon as follows:

Balance Sheet

Cash on Hand and in Bank:

Cash on hand was verified by actual count as at date of audit. Cash on deposit in General and Library Fund Accounts was verified by correspondence direct with The Toronto-Dominion Bank, Portage and Edmonton Branch, Winnipeg.

Fees Receivable:

This item represents 1958-59 membership fees unpaid at the year-end, as shown by the records of the Society. The unpaid balances have not been verified by correspondence with the members concerned.

Investments in Bonds:

The investments of the Society as at 30th April, 1959, were as follows:

	Cost	Par Value	Approximate Market Value
Government of Canada:			
3¾%, due 1978	$1,473.75	$1,500.00	$1,286.25
3¼%, due 1979	1,003.75	1,000.00	802.50
4½%, due 1983	3,031.88	3,000.00	2,793.75
The Hydro Electric Power Commission of Ontario:			
5%, due 1976	1,005.00	1,000.00	972.50
	$6,514.38	$6,500.00	$5,855.00

All of the above investments, held in a safety deposit box in The Toronto-Dominion Bank, were produced for our examination and in all cases are fully registered in the name of The Winnipeg Medical Society.

Statement of Revenue and Expenditure

The operations of the Society for the fiscal year resulted in net revenue of $748.43 on General Fund Account and net revenue of $569.13 on Library Fund Account. Full details are set forth on Exhibit B attached.

Revenue from membership fees is in accordance with your records, supported by duplicate receipts which were examined by us. All interest has been accounted for on a received basis.

All expenditures for the fiscal year have been approved in minutes of Council, and adequate vouchers were examined by us in support thereof.

* * *

We record with pleasure our appreciation of the courtesies and co-operation extended to us by Council members and staff during the course of our examination. Should any further information or explanations be required in connection with the attached accounts we shall be glad to be of service.

Auditors' Certificate

We have examined the balance sheets of The Winnipeg Medical Society and The Winnipeg Medical Society Library Fund as at 30th April, 1959, together with the related statements of revenue and expenditure for the fiscal year ended that date, and have obtained all the information and explanations we have required.

In our opinion, the attached balance sheets and statements of revenue and expenditure are properly drawn up so as to exhibit a true and correct view of the state of the affairs of the Society as at 30th April, 1959, and the results of its operations for the fiscal year then ended, according to the best of our information and the explanations given to us and as shown by the books of the Society.

Yours faithfully,
SILL, STREUBER & COMPANY,
Chartered Accountants.

EXHIBIT A

Balance Sheet
As at 30th April, 1959

ASSETS

	General Fund	Library Fund
Cash on Hand and in Bank	$2,060.71	$3,191.42
Membership Fees Receivable	70.00	
Investments — at cost:		
Government of Canada Bonds	5,509.38	
Hydro-Electric Power Commission of Ontario Bond	1,005.00	
	$8,645.09	$3,191.42

LIABILITIES

No Liabilities

Surplus:		
Balances as at 1st May, 1958	$7,896.66	$2,622.29
Net Revenue for fiscal year ended 30th April, 1959 (Exhibit B)	748.43	569.13
Balances as at 30th April, 1959	$8,645.09	$3,191.42
	$8,645.09	$3,191.42

EXHIBIT B

Statement of Revenue and Expenditure
For twelve months ended 30th April, 1959
General Fund

Revenue:		
Membership Fees:		
Active Members	$4,100.00	
Non-residents and Associates	14.00	$4,114.00
Bond Interest		251.24
		$4,365.24
Expenditure:		
Audit Fees	75.00	
Bank Charges, including Safety Deposit Box Rental	6.35	
Catering Expense	260.30	
Donations — Sundry	12.00	
Grant — Library Fund	1,000.00	
General Expenses	42.65	
Lantern Slides and Expense	50.00	
Manitoba Medical Association: Share of office salaries and expense	1,320.00	
Printing, Postage and Stationery	665.06	
Speakers' Expenses	178.35	
Telephone — Long Distance Tolls	7.10	3,616.81
Net Revenue for Year (Exhibit A)		$ 748.43

Library Fund

Revenue:		
Bank Interest	$ 43.78	
Grant — General Fund	1,000.00	$1,043.78
Expenditure:		
Books and Periodicals Purchased	$ 222.65	
Library Supervision	252.00	474.65
Net Revenue for Year (Exhibit A)		$ 569.13

Benevolent Fund

5th May, 1959.

To the Members,
The Winnipeg Medical Society Benevolent Fund,
Winnipeg, Manitoba.

We have examined the accounts of the Fund for the period 1st May, 1958 to 16th February, 1959, and submit the following statement for your consideration:

Balance of Fund, 1st May, 1958		$7,016.29
Add:		
Income during Period:		
Donations received	$ 50.00	
Bank Interest	116.00	
Bond Interest	22.50	188.50
		$7,204.79
Less:		
Expenses during Period:		
Safety Deposit Box Rental		4.00
Balance of Fund, 16th February, 1959		$7,200.79

As at 16th February, 1959, The Winnipeg Medical Society Benevolent Fund assets were transferred to The Manitoba Medical Association Benevolent Fund. The assets so transferred were as follows:

Cash on Deposit:		
The Toronto-Dominion Bank	$5,734.54	
Government of Canada Bonds:		
4½%, due 1983, at cost	1,466.25	$7,200.79

The transfer was made pursuant to resolution adopted at the Annual Meeting of the Members of The Winnipeg Medical Society held on 23rd May, 1958, authorizing the transfer.

Should any further information or explanations be required in connection with the foregoing statements, we shall be glad to be of service.

Yours faithfully,
SILL, STREUBER & COMPANY,
Chartered Accountants.

Benevolent Fund

To the President and Members of
The Winnipeg Medical Society:

For a number of years it had been considered advisable that the Winnipeg Medical Society benevolent fund broaden its scope and become a provincial wide organization. The time had finally arrived for action to be taken in this regard; accordingly the trustees of the Winnipeg Medical Society benevolent fund with the acquisence of the executive of the Winnipeg Medical Society made an offer of all its assets to the Manitoba Medical Association in order to set up a benevolent fund to include the medical profession of the entire province. At the 1958 annual meeting of the Manitoba Medical Association this offer was accepted. Complete transference of the assets of the Winnipeg Medical Society benevolent fund to the Manitoba Medical Association was concluded in February, 1959. These assets consisted of:

Bank Balance	$5,734.54
Government Bonds	1,500.00
	$7,234.54

The Winnipeg Medical Society Benevolent Fund began in May, 1947. The first meeting of its trustees took place May 8, 1947. It ceased to exist as an organized body in February, 1959.

Respectfully submitted,

David Swartz,
Chairman.

Community Chest

To the President and Members of
The Winnipeg Medical Society:

Herewith an account of the contributions from the Winnipeg doctors to the Community Chest for 1958.

This year 387 doctors and 132 employees contributed $22,808.50, which is 108% of the objective set by the General Campaign Chairman for this division. While it is very gratifying to have achieved in excess of the objective, it should be noted that only 73.7% of the doctors contributed, whereas a larger percentage of doctors contributed in previous years. Thus, larger contributions by fewer doctors accounted for the success of this year's campaign.

Of the 132 employees, 114 are in Clinics, and 18 were in individual doctors' offices.

Additional donations which cannot be calculated may have been by doctors through various organizations to which they contribute as members other than directly to the Red Feather solicitors. The same applies to employees of doctors.

In difficult cases of Diarrhoea

Many cases of diarrhoea in infants have been successfully treated without antibiotics—with Borden's Protein Milk.

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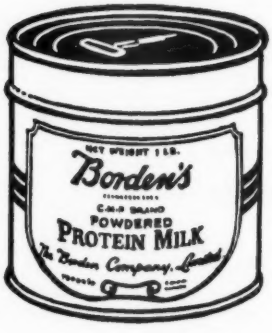

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The Chairman and Members of the Campaign Committee have extended their personal thanks together with the gratitude of the 40 Red Feather agencies for the continued support from th members of the medical profession. We in turn owe them a vote of thanks for undertaking the onerous responsibility of running the campaign and the actual convassing of our members.

The assistance of Dr. James Hart as a special assistant is gratefully acknowledged.

Respectfully submitted,

R. E. Beamish,
Representative.

Library

To the President and Members of
The Winnipeg Medical Society:

As your representative on the Medical Library Committee, I beg to submit the following report for the year 1958-59.

The medical library was fortunate in receiving a special grant from the University of $5,000.00 to be used in binding, and this has been of great help in making an impression on the accumulated mass of unbound journals. This grant has been expended, and it is planned to use the Winnipeg Medical Society grant in binding about 70 volumes more.

Dr. Ormerod, the Chairman of the Medical Library Committee, has been ill, and the spring meeting of the Book Selection Committee was therefore postponed. However, it is plannd to spend approximately $500.00 for references, texts, and other new titles.

The following is a brief summary of activities and expenditures which was prepared by Miss Cynthia Roblin, Librarian, for the Society's information:

EXPENDITURES

Books — 60 to 70 titles	$500.00*
Binding — 65 to 70 volumes	500.00*
Evening hours — 18/2/59 to 16/5/59	236.00
	$1,236.00

*Estimated.

BOOK DISPLAYS

Book displays presented before six meetings.

BORROWERS

Registered physicians in Winnipeg	736	
Registered borrowers	258	(35%)
Number of items borrowed	8,247	

Respectfully submitted,

J. T. MacDougall,
Representative.

Membership

To the President and Members of
The Winnipeg Medical Society:

Membership in the society is as follows:

Active paid-up members		382
New members	29	
Active paid-up members (half rate)		42
Associate or Non-resident members		7
Total paid-up members		431
Life membership		31
Free membership (over 65)		28
Non active		7
Membership fees unpaid		7
		504

This represents a net increased membership of 15 in spite of a loss of 10 members in the past year — 5 members have died and 6 have moved from the district). The "membership fees unpaid" category has been increased from 3 to 8.

Respectfully submitted,

G. A. Waugh,
Chairman.

Legislative

To the President and Members of
The Winnipeg Medical Society:

During the past year several matters of Legislation affecting the Medical Profession directly or indirectly were brought before the Provincial House. As these matters had medical implications beyond the restricted sphere of the Winnipeg Medical Society they were dealt with by the M.M.A. Consequently, no meetings of this committee were held during the past year.

Respectfully submitted,

A. B. Houston,
Chairman.

Program

To the President and Members of
The Winnipeg Medical Society:

During the year 1958-1959 the following meetings of the Winnipeg Medical Society were held. The titles and speakers were as follows:

SEPT. 26, 1958
"Common Congenital Anomalies"
Dr. Benjamin K. Rank, Melbourne, Australia.

OCT. 24, 1958
"Impending Myocardial Infarction — Recognition and Management — Observations on the Mode of Action of Oral Anticoagulants"
Dr. Robert E. Beamish, Winnipeg.

NOV. 21, 1958
"Early Surgeons of the North West Mounted Police"
Dr. John B. Ritchie, Regina, Saskatchewan.

DEC. 5, 1958
"Experience in Treatment of Surgical Lesions of the Pancreas"
Dr. Jonathan E. Rhoads, Pennsylvania.

JAN. 23, 1959
The General Practitioners' Association of Manitoba.
"Trauma — Aspects of Highway Traffic Trauma"
Dr. W. S. Reid, Selkirk.
"Consideration of the Oesophagus"
Dr. R. A. Jacques, St. Boniface.
"The Precious Commodity"
Dr. Athol Gordon, Winnipeg.

FEB. 20, 1959
"The Annual Clinical Session"
Winnipeg General Hospital.

MAR. 20, 1959
"Medical Problems in the Arctic"
Dr. J. A. Hildes, Winnipeg.

APRIL 24, 1959
The Clinical Significance of Epileptic Seizures
Dr. G. L. Adamson
Dr. R. T. Ross
Dr. M. G. Saunders.

It is a pleasure to thank the Executive of the Winnipeg Medical Society for their help during the year and to thank the speakers for their excellent papers.

Respectfully submitted,

T. W. Fyles,
Chairman.

Public Health

To the President and Members of
The Winnipeg Medical Society:

As there were no meetings of the Standing Committee on Public Health of the Winnipeg Medical Society, there is nothing to report.

Respectfully submitted,

R. G. Cadham,
Chairman.

Public Relations

To the President and Members of
The Winnipeg Medical Society:

There has been continued incidence of members notifying your committee of various talks to other than medical audiences.

Respectfully submitted,

M. McLandress,
Chairman.

Trustees

To the President and Members of
The Winnipeg Medical Society:

As senior Trustee, I wish to report the following securities as being held in the Safety Deposit Box, Toronto-Dominion Bank, 394 Portage Avenue, as inspected by Doctors J. A. Swan and D. L. Kippen:

Three Government of Canada Bonds, 4½% maturing 1983 — each $1,000.00	$3,000.00
Three Government of Canada Bonds, 3¾% maturing 1978 — each $500.00	1,500.00
One Government of Canada Bond, 3¼% maturing 1979 — at $1,000	1,000.00
One Hydro Electric Power Commission of Ontario Bond, 5% maturing 1976 — at $1,000.00	1,000.00
	$6,500.00

Your Trustees took advantage of the Canadian Government's conversion offer on Victory Bonds held by your Society and authorized conversion of $3,000.00 worth of 9th Victory loan bonds to $3,000.00 worth of 4½% 25-year Government of Canada Bonds.

Respectfully submitted,

John A. Swan,
Trustee.

Welfare Council

To the President and Members of
The Winnipeg Medical Society:

Your representative attended most meetings of the Health Division of the Welfare Council of Greater Winnipeg. Again the only general meeting of the Welfare Council of Greater Winnipeg was the annual meeting which was reported in detail in the local Press at the time. The highlight of this meeting was an address by Archbishop Pocock, both timely and interesting but of no special medical interest.

The Health Division held several luncheon meetings through the year. It also held a mid morning "Coffee Party" at which all members of the division were presented to the new Minister of Health, Dr. George Johnson. Mr. Gordon Pickering, the new Commissioner of Hospitalization, also addressed the division on another occasion.

Your representative received a request from the Welfare Council that the Winnipeg Medical Society endorse their report on psychiatric services in Greater Winnipeg. It was recommended that further information regarding this survey should be submitted and the report passed to the psychiatric section for their comment. This comment has not, as yet, been received.

Respectfully submitted,

P. K. Tisdale,
Representative.

Anaesthesia

To the President and Members of
The Winnipeg Medical Society:

It gives me great pleasure to submit a report on the activities of the Winnipeg Anaesthetists' Society for the 1958-59 season.

JULY, 1958

A Professional Policy Committee of the Winnipeg Anasthetists' Society was formed. This committee, the name of which was subsequently changed to the Anaesthetic Planning Committee, was comprised of one representative from each hospital and was to last for a period of two years. The programme for the 1958-59 season was drawn up on July 3rd.

OCTOBER, 1958

Dr. P. Bromage of Montreal spoke to the group on some aspects of compliance and ventilation and epidural anaesthesia as applied to the relief of intractable pain.

NOVEMBER, 1958

Two papers were presented: one on Epidural anaesthesia by Dr. Kantor and the other on the Control of Haemorrhage in Tonsillectomy by Dr. Erenberg.

DECEMBER, 1958

The annual Christmas Dinner-Dance was held in the Club Morocco.

JANUARY, 1959

The first of the two papers presented was that of the Children's Hospital under the joint authorship of Drs. T. McCaughey, H. Reid and M. Saunders who discussed at some length "Cardiac Arrythmias in Squint Surgery." The second paper "A Review of Hypothermia with a description of its use in two cases" was presented by Dr. Camrass, representing St. Boniface Hospital.

FEBRUARY, 1959

The topics presented at this meeting were: "Hiccup" by Dr Nip, Resident in Anaesthesia at Deer Lodge Hospital and "T.U.R. and the Anaesthetist" by Dr. Cham of Victoria Hospital.

MARCH, 1959

At this meeting the General Hospital was responsible for the scientific portion: thus Dr. Wolkenstein spoke on "Anaesthesia after Dibenzyline had been administered for shock due to trauma" while Dr. McCammon spoke on the Mayo Clinic with particular reference to the Anaesthetic Department.

APRIL, 1959

It is proposed for this meeting that interesting cases be presented, whilst for the final meeting in MAY we hope to have as guest speaker, Dr. Robt. Smith from Boston who proposes to speak on "Fluid Balance and Mortality in Paediatric Anaesthesia."

Respectfully submitted,

J. Crawford,
Secretary-Treasurer.

General Practice

To the President and Members of
The Winnipeg Medical Society:

The following is a summary of the activities of the General Practitioners' Association of Manitoba for the year 1958-1959. The Executive of this association met on the third Tuesday of each month.

The Annual Dinner Meeting was held on October 6th, 1958. A special general meeting was held on December 16th, 1958, to discuss the formation of a Section of General Practice of the Manitoba Medical Association. To conform with the new constitution of the M.M.A., those present at the meeting voted to fill an application for recognition as the Section of General Practice of the M.M.A., and to retain the General Practitioners' Association as an affiliated body. This application has been approved by the Executive of the M.M.A.

The Annual Valentine's Dinner and Dance was held on February 14th and was again a very enjoyable and successful occasion.

The meeting of the Winnipeg Medical Society of January 23, 1959, was chaired by Dr. D. J. Hastings, and a very interesting and informative program was presented by Dr. W. S. Reid, Dr. Athol R. Gordon, and Dr. R. A. Jacques.

Respectfully submitted,

A. A. Campbell,
Secretary.

Internal Medicine

To the President and Members of
The Winnipeg Medical Society:

During 1958-59 session, this section held four general meetings. These meetings were well attended. The section heard and discussed the following papers:

1. The significance of abnormal E.C.G.'s in apparently normal people — Dr. F. A. L. Mathewson.
2. The Patho-Physiology of Basal Nuclear Sessions — Dr. Burdon Martin, London, England.
3. Obesity — a psychiatrist's observation on one obese person — Dr. Brian Bird.
4. Review of the Cardiology Congress, Brussels — Dr. F. A. L. Mathewson and Dr. R. Beamish.

Respectfully submitted,

J. F. S. Hughes,
Secretary.

Medical History

To the President and Members of
The Winnipeg Medical Society:

This section held three meetings during the 1958-59 season at the Medical Arts Clubrooms, each preceded by a dinner.

The November Meeting was addressed by Dr. Ritchie of Regina. He gave a very scholarly discourse on the Medical Practitioners associated with the Royal Canadian Mounted Police during the early history of Western Canada. This meeting was attended by members of the Medico-Legal Society as well as by members of the Medical Society.

Dr. Neilson, the Dean of our new School of Dentistry, addressed the January meeting with a very fascinating talk on Dental History.

The March meeting was addressed by Dr. Sigurdson, presenting a very interesting account entitled, "Medicine in Mexico," going back to the time of the Spanish conquerors and on up to the present.

Dr. Mitchell has tendered his resignation as President of the Section and this was accepted with deep regret. Dr. L. A. Sigurdson was elected as President for the coming year.

Respectfully submitted,

Dwight Parkinson,
Secretary.

Obstetrics and Gynaecology

To the President and Members of
The Winnipeg Medical Society:

Three meetings of the Section were held in the past year. At a dinner meeting, Professor T. N. A. Jeffcoate presented a paper on "Stress Incontinence."

Respectfully submitted,

C. C. Henneberg,
Secretary.

Paediatrics

To the President and Members of
The Winnipeg Medical Society:

The Pediatric Section held a dinner meeting in honour of Dr. Horace Hodes on December 4, 1958, with 27 present. Dr. Hodes spent a week in Winnipeg as visiting Professor of Pediatrics. On March 21, 1959, a meeting was held to hear a report from Dr. S. Israels regarding Professional Policy Committee.

Respectfully submitted,

Maurice Berger,
Secretary.

Radiology

To the President and Members of
The Winnipeg Medical Society:

During the year the Section of Radiology of the Winnipeg Medical Society met on four occasions to discuss matters of current interest. The officers elected this year are — Chairman, Dr. W. J. Elliott; Secretary, Dr. J. B. Squire; Treasurer, Dr. C. W. Hall.

Respectfully submitted,

J. B. Squire,
Secretary.

Eye, Ear, Nose and Throat

To the President and Members of
The Winnipeg Medical Society:

During the year 1958-59 this Section has had two general meetings and one special meeting.

Dr. P. G. Martens spoke on "Divergent Strabismus." A second regular meeting took place at which Dr. Guest and Dr. McGoey discussed the American Academy of Ophthalmology and Otolaryngology meeting. A special meeting was held with Miss Hickling of the Toronto National Institute for the Blind, with regards the establishment of an eye bank in Winnipeg.

Respectfully submitted,

R. C. Davison,
Secretary.

Executive Committee, M.M.A.

To the President and Members of
The Winnipeg Medical Society:

As representative to the Executive Committee of the Manitoba Medical Association I have attended all regular meetings and reported all pertinent information back to the Council.

Respectfully submitted,

Dwight Parkinson,
Secretary.

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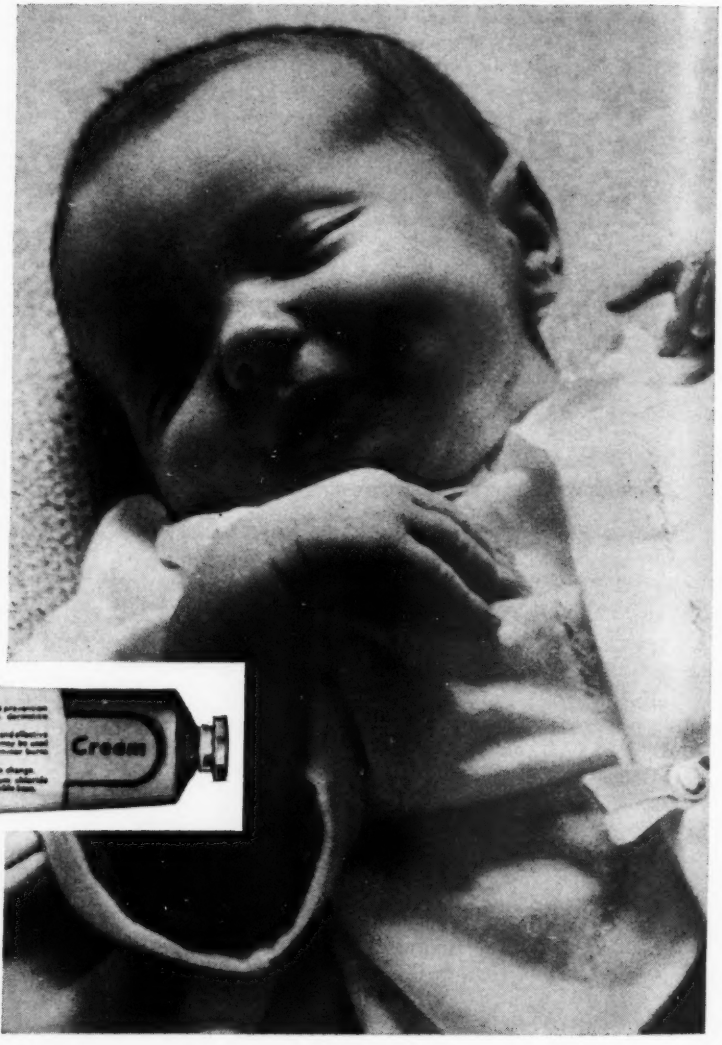

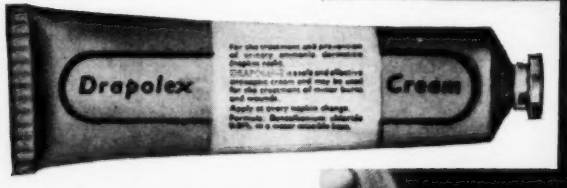

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HOSPITAL MEETINGS

Hospital	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
Children's	12:00 Surgical Rounds	10:00 Staff Rounds "B" Service 12:00 Postgraduate Seminars	9:30 Staff Rounds "C" Service 11:00 Clinical Path. Conference 11:00 Death Review (4th Wednesday)	11:00 Grand Ward Rounds	10:00 Staff Rounds "A" Service 12:30 Clinical Luncheon (1st Friday)	9:00 Newborn Conference
Deer Lodge	Clinical Luncheon (1st Monday)			11:00 Ward Rounds		
Grace		Clinical Luncheon (3rd Tuesday)	9:00 Paediatrics 9:45 Medicine 10:30 Ob. & Gyn. 11:15 Surgery	12:00 - 2:00 p.m. Weekly Seminar		
Misericordia	Tissue Committee (3rd Monday)	Clinical Luncheon (2nd Tuesday)				8:30-12:30 Ward Rounds
Municipal			7:30 p.m. Review of Deaths (2nd Wednesday)			8:30 Clinical Staff Conf. and Ward Rounds
St. Boniface	11:00 Paediatric Rounds	11:00 Surgical Rounds	9:00 Obstetrical 11:00 Grand Rounds 12:30 Cardiac Unit (4th Wednesday)	11:00 Tumor Clinic 12:00 Clinical Luncheon (2nd & 4th Thurs. ex. July & Aug.)		
St. Boniface Sanatorium	12:30 Clinical Luncheon (1st or 2nd Monday)					
Victoria					Clinical Luncheon (3rd Friday)	
Winnipeg General		11-12:00 Medical Ward Rounds	9-10:00 Tumor Clinic 10-11:00 Surgical	12:15 Clin. Lunch. (1st & 3rd Thurs.)		
Brandon General			Medical Staff Lunch (Wed. prior to 2nd Tues. each month)			10:00-noon Clinical Drs. from S.W. Manitoba invited (Nov. to May)

Detailmen's Directory

Representing Review Advertisers in this issue, whose names are not listed under a business address.

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DEPARTMENT OF HEALTH & PUBLIC WELFARE

COMMUNICABLE DISEASE PICTURE

North of 53 District

Bacillarydysentery, infectious hepatitis and pertussis head diseases reported. Scarlet fever is on the decrease.

Northern District

Scarlet fever very prevalent throughout the entire district. Several cases of infectious hepatitis and bacillary dysentery reported.

Northwestern District

Bacillary dysentery still being reported but no other diseases of any significance.

Brandon District

Infectious hepatitis still prevalent with scarlet fever decreasing. Three new cases of tuberculosis reported.

Central District

No significant changes over last month. Scarlet fever and tuberculosis cases head the list.

Winnipeg District

Scarlet fever cases reported show no decrease, but infectious hepatitis cases are decreasing. One case of proven diphtheria reported but no further spread. A few cases of enteritis under four weeks of age are now being reported.

Southern District

Scarlet fever is the only infectious disease reported for the month from this area.

LIST OF DEATHS FROM COMMUNICABLE DISEASES
April, 1959

URBAN: Cancer, 72; Influenza, 19; Jaundice (infectious), 2; Meningitis (meningococcal), 1; Pneumonia, Lobar (490), 6; Pneumonias (other forms), 38; Septicaemia and Pyaemia, 1; Syphilis, 2; Tuberculosis, 2; Other Bacterial diseases, 1. Other deaths under 1 year, 27. Other deaths over 1 year, 275. Stillbirths, 10. Total, 456.

RURAL: Cancer, 36; Diarrhoea and Enteritis, 2; Influenza, 10; Pneumonia, Lobar (490), 2; Pneumonias (other forms), 10; Septicaemia and Pyaemia, 1; Tuberculosis, 2. Other deaths under 1 year, 13. Other deaths over 1 year, 186. Stillbirths, 12. Total, 274.

INDIANS: Cancer, 1; Diarrhoea and Enteritis, 1. Other deaths under 1 year, 1. Other deaths over 1 year, 4. Total, 7.

General

Influenza is definitely on the wane in greater Winnipeg and appears to be declining in rural Manitoba. Most reports of "flu" are coming in from the west and northern parts of the Province.